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ACCIDENTAL POISONING IN CHILDREN IN CAPE TOWN, WITH SPECIAL REFERENCE TO KEROSENE AND SALICYLATES

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The total number of cases of poisoning admitted to the childrens' wards at Groote Schuur Hospital, Observatory, Cape, during the years 1951 - 1958 inclusive, and to the Red Cross War Memorial Childrens' Hospital, Rondebosch, Cape, from the time of its opening in June 1956 to the end of 1958, was small when compared with other reports, especially those from Johannesburg.[1,2] However, many of the Cape Town cases were treated as out-patients and not admitted to the wards.

There were 127 cases at Groote Schuur Hospital during the 8-year period, being 1·8% of admissions to the childrens' medical wards. There were 80 European, 39 Coloured, and 8 African children. These represented 3·3%, 1·0% and 1·0% of the total admissions for the respective racial groups. Three children died, a mortality of 2·4%.

The Red Cross War Memorial Childrens' Hospital had 39 cases in 2½ years, 18 being European, 17 Coloured and 4 African. The figure is 1·4% of the total admissions to the medical wards during this time. By races, the percentages of all admissions were 2·0%, 1·3% and 0·8% for European, Coloured and African children respectively.

Craig and Fraser[3] reported a continual increase in the number of cases of poisoning in children in 2 Scottish hospitals over a 20-year period up to 1951, and the Johannesburg workers' figures indicated a similar trend. The Groote Schuur Hospital figures for the period under review did not, however, show any such increase.

It is noteworthy that in our series there were considerably more Europeans than non-Europeans, despite the fact that of the patients admitted for all causes, about 66% were non-European. This is also reflected in the figures for children presenting with poisoning at the casualty department of Groote Schuur Hospital during 1958. In this period there were 39 Europeans and 29 non-Europeans. This does not, of course, necessarily mean that there were more cases of poisoning among Europeans in the community as a whole, since it may be that non-European children are not always brought to hospital unless symptoms are severe.

TABLE I. AGE INCIDENCE OF ACCIDENTAL POISONING

Hospital	*Dates*	*0 - 1 Year* %	*1 - 2 Years* %	*2 - 5 Years* %	*Over 5 Years* %
Cape Town	1951-1958	5	49	36	10
Scottish	1931-1951	5	37·4	47	10·6
Johannesburg (1)	1948-1952	6·4	43·7	39·1	10·8
(2)	1953-1955	6·8	48·2	40	10

Age Incidence

Our highest incidence was in the 1 - 2 year group, followed by the 2 - 5 year group. These figures agree with those of the Johannesburg workers,[1,2] while Craig's figures showed the opposite.[3] In the groups under 1 year and over 5 years all 3 centres had similar figures (Table I).

Nature of the Poison

Table II shows the commonest poisons ingested in order of frequency in the Cape Town, Johannesburg and Scottish series and in the childrens' section of Addington Hospital, Durban, as reported by Simson.[4]

In all 3 South African centres *kerosene* was the poison most frequently taken by children. *Barbiturate* poisoning was fairly common in all the reports quoted, but was seldom fatal. All our cases in this group recovered rapidly, though there was often marked ataxia for a few days. In only 1 case was it thought necessary to give bemegride (a reputed respiratory stimulant), but since its efficacy is doubtful, it will probably not be used in future cases.

Arsenical poisoning seems to be relatively more common in Cape Town than in the other 3 centres. We had 19 cases, mostly from the ingestion of ant-poison, but in a few instances 'sluggem' pellets, which contain metaldehyde as well as arsenic, had been taken. Full recovery followed in all but 2 of these patients, 1 dying shortly after admission. In the other case the child developed convulsions some months later, which may have been related to the previous arsenical ingestion.

TABLE II. FREQUENCY OF COMMONER POISONS

Poison	*Cape Town 1951-1958*	*Johannesburg 1948-1952*	*Johannesburg 1953-1955*	*Scottish hospitals 1931-1951*	*Durban 1949-1953*
Kerosene	43	152	119	31	42
Barbiturates	21	36	28	36	7
Arsenic	19	16	24	—	8
Salicylates	14	82	104	20	6
Caustic soda	3	57	17	4	9
Disinfectants	—	—	—	39	9
Camphor liniment	—	11	9	29	2
Turpentine	—	8	6	25	—
Ferrous sulphate	4	1	6	24	1
Total cases seen	166	594	527	296	126

Salicylate poisoning is seen quite often, and may carry a high mortality, especially when the drug is given in repeated doses to a febrile, dehydrated child. The high salicylate content of oil of wintergreen is well known,

about 45 gr. of salicylate per fluid drachm, but it is doubtful whether the public is aware of its lethal qualities.

A great many cases of *ferrous sulphate* poisoning, with a fairly high mortality rate, have been reported, especially from Great Britain. We had only 4 cases, all with minor symptoms and all recovering rapidly. The Johannesburg authors had only 1 case in their first report and 6 in their second.

TABLE III. TYPE OF POISONING AT GROOTE SCHUUR AND RED CROSS WAR MEMORIAL CHILDREN'S HOSPITALS (1951 - 1958)

	Eur.	*Col.*	*Afr.*	*Total*
A. Medicinal drugs:				
Barbiturates	14	7	0	21
Salicylates	10*	4	0	14
Sulphonamides	3	1	0	4
Ferrous sulphate	1	3	0	4
'Largactil'	0	3	0	3
Digitalis	3	0	0	3
Glyc. trinitrate	2	0	0	2
'Dexedrine'	2	0	0	2
Chloral	1	1	0	2
'Theominal', veg. lax., antihistamine, hyoscine—1 of each	4	0	0	4
INH	0	1	0	1
	40	20	0	60†
B. Household preparations:				
Kerosene	17	20*	6	43
Other hydrocarbons	4	2	0	6
Arsenic	12	7*	0	19
Insecticides	4	1	0	5
Caustic soda	2	0	1	3
Other corrosives	1	1	0	2
Alcohol	0	1	2	3
Antiseptics	3	0	0	3
Solder fluid (ZnCl)	2	0	0	2
Others, 1 each	6	1	0	7
	51	33	9	93†
C. Miscellaneous:				
Seeds	3	3	0	6
Belladonna (plant)	3	0	0	3
Scorpion bite	1	0	0	1
Snake bite, CO, and unknown poison—1 each	0	0	3	3
	7	3	3	13†

* One death in each of these groups.
† Grand total: 60 + 93 + 13 = 166.
Eur. = European, Col. = Coloured, and Afr. = African.

Table III gives the complete list of poisons for which the children were admitted to the 2 Cape Town hospitals.

KEROSENE POISONING

This was by far the commonest form of poisoning in the Cape Town hospitals, there being 43 cases in all. Seventeen were in European and 26 in non-European children.

Pulmonary Involvement

X-ray evidence of pulmonary involvement was very frequent, 27 of the 32 Groote Schuur Hospital cases showing infiltration of some part of the lung fields. The basal zones were always involved, commonly bilaterally. In a few of the more severe cases the hilar regions were also affected, but in none of the films did the upper lobes appear to be involved. That the changes occur early was shown by the fact that, in many of the patients who underwent X-ray examination in the casualty department soon after ingestion of the kerosene, infiltration was already apparent.

Clinical evidence of pulmonary involvement was present in the majority of cases, either at the time of admission or later, but some did not at any time have abnormal physical signs despite radiological changes. Although clinical recovery was rapid in most cases, the radiological signs tended to persist for some time. The average duration of stay in hospital was 9 days. Fever was present in more than 70% of patients, and a leucocytosis of over 10,000 per c.mm. was found in 17 out of 22 cases where a leucocyte count was recorded.

Nearly all patients underwent gastric lavage in the casualty department before admission to the wards. There is no proof that any became more ill because of this, but the procedure is unnecessary and, as is well known, lavage may be followed by regurgitation and further aspiration of kerosene.

How Does the Kerosene Reach the Lungs?

The usual story in our cases was the well-recognized one that on taking the kerosene the child immediately began to choke and cough — this direct aspiration of kerosene is the generally accepted route. It is, however, often stated that kerosene is also absorbed from the stomach and excreted into the alveoli through the pulmonary capillaries.

This view was advanced by Deichmann *et al.*[5] These workers introduced kerosene directly into the stomachs of several rabbits, and into the peritoneal cavities of others. They stated that on performing autopsies they found pulmonary changes, similar to those found after kerosene had been inhaled, in both groups of animals. Because of these findings they recommended stomach aspiration in cases of kerosene poisoning.

On the other hand, Lesser *et al.*[6] reported contrary results in similar experiments. Kerosene was run into the stomachs of 6 rabbits, but none of these showed any radiological or autopsy evidence of lung pathology. In 6 other rabbits, intratracheal instillation of kerosene resulted in both X-ray changes in the lungs and autopsy signs of oedema of the air passages with haemorrhages into the lung parenchyma. This work confirmed similar studies by Waring,[7] and by Reed *et al.*[8]

In 1954 Foley *et al.*[9] tied off the oesophagus in 10 rabbits and passed stomach tubes through openings below the ligatures. Kerosene was poured down the tubes and the animals were killed. Postmortem examination showed intense congestion of the brain but no lung changes either gross or microscopic. On the other hand, kerosene run down the trachea produced the same changes as found by others, namely haemorrhagic, necrotizing bronchopneumonia and 'asphyxial membranes' lining the alveoli.

Although there has been some experimental support for Deichmann and the matter cannot be said to be finally settled, it is evident that most workers favour aspiration as the sole cause of the lung pathology.

A point which probably supports aspiration is the finding, in our cases, and apparently in others also, that the upper zones of the lung fields always seem to be much less affected than the basal areas. This is contrary to what one would expect if there was a blood-borne carriage of kerosene to the lungs. Foley *et al.*[9] support this view.

In summary, kerosene is a very common form of poison-

ing in young children, although the mortality is low. X-ray changes in the lung fields occur in nearly every case even when there is no clinical evidence of pulmonary involvement. The respiratory pathology is probably solely the result of aspiration, and gastric lavage is to be deprecated.

SALICYLATE POISONING

This is one of the commonest forms of poisoning in the USA and elsewhere, and the many reports appearing on the subject indicate the magnitude of the problem.

Symptoms

Young children usually do not complain of the early symptoms of tinnitus, deafness and giddiness, and the most striking feature of salicylism in children is hyperventilation. There may also be fever, sweating, thirst and vomiting, all leading to dehydration. The child is often restless and drowsy and may become cyanosed. This may be followed by coma, convulsions and, finally, death from respiratory failure. There may be bleeding, particularly from the stomach or the nose, or into the skin, and intracranial bleeding has also been reported. This is usually due to hypoprothrombinaemia, but may rarely be the result of thrombocytopenia. Another complication which is mentioned by Hill[10] is a toxic encephalopathy.

The symptoms may suggest encephalitis, bronchopneumonia or diabetic ketosis, but the cerebrospinal fluid and lungs are found to be normal. Diagnostically, it is important to think of the possibility of salicylism in a child who is hyperventilating. The ferric chloride test will show the presence of salicylates in the urine and the serum-salicylate level may be high. This level, however, is variable, and Heymann *et al.*[11] reported cases from Johannesburg where symptoms were present with levels as low as 11 mg. per 100 ml. On the other hand, some of their patients had salicylate figures of 50 mg. per 100 ml. without symptoms.

Heymann's article on salicylate intoxication[11] discussed the problem fully and what follows is largely an amplification of that review, particularly with regard to the biochemical disturbance and treatment in the light of present-day opinion. Salicylate poisoning is so important that it seems worth while reiterating many of the points which Heymann made.

Mortality

This may be high, especially when a child receiving salicylates is already sick with fever, vomiting, poor fluid intake, and accompanying oliguria; the duration of the oliguria has an important bearing on the outcome of the case. If a healthy child swallows a quantity of salicylate the prognosis is usually better. This was well shown in Heymann's series.

Local Experience

In the 2 Cape Town hospitals there were 14 cases of salicylate poisoning, 10 of the children being Europeans. In nearly all of them the drug was self-administered and most recovered within a few days without trouble. Two cases, however, will be briefly described.

In the first, a child, aged 14 months, had been given intermittent doses of 'disprin' over a few days for a febrile illness. For 1 day before admission he had been restless and drowsy and had vomited all feeds. On admission he was hyperventilating and comatose. The serum salicylate level was 80 mg. per 100 ml., the CO_2 was 19 volumes %, and the blood urea 70 mg. per 100 ml. Under treatment he became hyperpyrexial and developed generalized convulsions. He improved physically but his final mental state was one of gross retardation, even allowing for the fact that he was said to have been somewhat backward in the past. There remained some uncertainty whether there was not in addition an encephalitis, as he had had measles a few weeks earlier. Alternatively, this may have been an example of the toxic encephalopathy mentioned by Hill.[10]

In the second case, a child aged 18 months was said to have swallowed about 1 oz. of oil of wintergreen. After stomach lavage the parents were allowed to take the baby home as there appeared to be no symptoms of poisoning but, on admission to the ward the next morning, she was cyanosed and very dyspnoeic, and rapidly became comatose. Convulsions followed, and the temperature rose to 106°F. Death occurred about 8 hours after admission despite treatment. At autopsy a very congested brain, with a subdural haematoma, was noted.

Pathology and the Biochemical Disturbance

It is thought that the first effect of salicylate is direct stimulation of the respiratory centre, resulting in hyperventilation and a respiratory alkalosis which may be severe enough to give rise to tetany. Corrective renal mechanisms come into play which bring about an increase in plasma chloride and decrease in bicarbonate and tend to reduce the alkalaemia. This may take some time, but there is probably some immediate buffering action by the haemoglobin and very likely by other mechanisms as well.

The result of all this is a change to a metabolic acidosis, which is enhanced by the acid salicylate still circulating and by the impaired renal function. Salicylates are slowly excreted even when renal function is normal and under the conditions mentioned above, excretion is even slower. The pre-existing state of dehydration and starvation in the sick child, together with the circulating salicylate, bring about a disturbance of carbohydrate metabolism with the development of ketosis, much as occurs in diabetic precoma. It would seem that salicylate interferes with several systems involved in carbohydrate metabolism.

A recent article by Winters and others[12] suggests that the position is even more complicated than outlined above, at any rate in infants and very young children. In 33 patients in this age group they found a simultaneous disturbance involving both a primary respiratory upset, shown by hyperventilation, and a primary metabolic disturbance, shown by ketosis. In other words, there appear to be several disturbances all operating simultaneously and the blood pH may be alkaline, acid or neutral. In older children and adults, however, the sequence seemed to be as outlined earlier.

Another toxic effect of salicylate is increase of the metabolic rate and heat production, presumably in skeletal muscle. If this effect exceeds the antipyretic effect of the drug, the heat loss from sweating and hyperventilation may be insufficient to control the patient's temperature, and hyperpyrexia may result.

Hyperventilation persists for some time and, in fact, prevents an extreme fall in blood pH. Stimulation of the respiratory centre by salicylate continues until the salicylate blood level has fallen considerably. Overbreathing, therefore, is not a good indication whether the disturbance is one of alkalosis or acidosis, and estimation of the blood pH is the only way to be sure of this since the CO_2 figures may well be low in either state.

The dehydration which occurs is said to be more the

result of water loss than of electrolyte depletion, and hypernatraemia may develop unless the relative excess of electrolytes can be removed. The volume of urine excreted may be insufficient to achieve this and the serum will then remain hyperosmolaric. Serum potassium is, however, sometimes decreased. The explanation for this is not clear, unless salicylate stimulates the pituitary gland to produce corticotrophic hormone with consequent potassium loss from adrenocortical activity.

Although laboratory findings may be somewhat variable, in the later stages, at any rate, they are likely to show a lowering of the following — CO_2-combining power, pH, prothrombin and possibly potassium; and an increase of sodium, urea and sugar.

TREATMENT OF SALICYLATE POISONING

Since there often is a delay in onset of symptoms, as in our second case, all patients attending hospital after ingesting salicylate should be admitted for observation.

The Rôle of Lavage

Washing out the stomach in cases of poisoning is a time-honoured form of treatment. Arnold *et al.*,[13] however, point out that since 1900 there has been only 1 report of an attempt to assess its value. This was done by Scandinavian workers who stated that in 80 cases treated in this way insignificant amounts of poison were recovered. It was felt that much of the poison had already passed beyond the stomach but that, in addition, a significant amount was washed into the intestines during the act of lavage.

To test this Arnold and his co-workers introduced salicylate into the stomachs of dogs and found that if lavage was delayed by 1 hour, little was recovered, but if done early, about 38% could be washed out. Using syrup of ipecacuanha as an emetic, they were able to recover 45% of the poison if the ipecacuanha was administered early, and 39% if late.

It would seem, therefore, that emesis, even if late, is as effective as early lavage. Not everyone would care to use this method, however, and the authors agreed that where there is severe depression of the central nervous system emesis is contra-indicated. Their recommendation was that if lavage is used, it should be preceded by aspiration to avoid driving the poison further along the gastro-intestinal tract. Bicarbonate solution should not be used as this may facilitate the absorption of salicylate. In any event, if hyperpnoea is already present, it is too late for lavage.

The Threat to Life

The immediate threat to life comes from hyperpyrexia and dehydration, and the initial treatment must be to correct these by efficient methods of cooling and by copious fluids, in most cases given intravenously.

An adequate fluid to use is $\frac{1}{2}$-strength Darrow's solution in $2\frac{1}{2}$% dextrose-water. Once laboratory reports are to hand, electrolytes may be further adjusted if necessary. The amount of fluid as suggested both by Segar and Holliday[14] and by Winters[15] is 20 - 50 ml. per kg. body weight for rehydration, in addition to the usual amounts for maintenance according to body weight. These will vary from 150 ml. per kg. body weight in infants under 1 year of age to 80 ml. per kg. body weight between 4 and 10 years. If dehydration is severe it may be necessary to give up to 100 ml. per kg. body weight for rehydration. It is difficult to give a hard and fast rule regarding the speed at which fluid should be given. To re-establish renal flow, intravenous fluids can be given quite rapidly for the first 6 - 8 hours. Some caution must, however, be exercised, since too rapid hydration has been followed in some instances by pulmonary oedema.

Alkalis or Not?

There is considerable controversy on this point. It was emphasized by Heymann[11] and also in a commentary in *Pediatrics*[16] that large amounts of alkali may easily throw the patient back into alkalosis. In *Pediatrics*, the writer stated that the hazard from alkalosis is often greater than the modest degree of acidosis being treated. A pH of less than 7·15 probably demands alkali therapy, but this must be strictly controlled by serial pH and CO_2 studies. It was stated that in most cases specific therapy against alkalosis is probably not necessary. Segar and Holliday[14] held the same views and stated that restoration of hydration and the addition of electrolytes will rapidly ameliorate the metabolic acidosis and restore renal function, with the resultant excretion of the various accumulated organic acids.

Winters[15] found that patients, properly hydrated and given the necessary electrolytes and carbohydrate but little or no alkali, usually began to recover. This recovery was, however, associated with a significant tendency towards the development of alkalosis, and this tendency was both aggravated and accelerated when large amounts of alkalinizing salts were given. He nevertheless recommended intravenous bicarbonate if the pH was 7·15 or less, but demanded pH and CO_2 estimations immediately after such infusion.

Alkalinization of the urine is known to increase the rate of excretion of salicylate, and 'diamox' has been tried by some workers with this in view. Some benefit seemed to result but convulsions occurred in certain patients and the wisdom of this form of treatment seems very doubtful.

Whitten *et al.*,[17] reporting to the American Pediatric Society recently, felt that the risk of accentuating the respiratory alkalotic phase by the use of alkalis, as many have claimed, was largely theoretical or based on only a few animal studies. They used intravenous alkalis in 21 children suffering from salicylate intoxication, whose ages ranged from 4 months to 4 years. There was full laboratory control throughout, and the authors claimed that the rate of excretion of salicylate and consequent decrease in its serum level was comparable with the cases treated by dialysis or exchange transfusion. It was in the paper by Heymann *et al.*[11] that exchange transfusion for salicylate poisoning was first reported. Since then Done and Otterness,[18] as well as others, have recommended this for severe cases.

Extracorporeal Dialysis

Another method of ridding the body of salicylate is by extracorporeal dialysis, but it has been said that this is impracticable in small children for technical reasons. It was interesting, therefore, to see a recent report by Spritz *et al.*[19] on the successful treatment of a 13 kg. boy using one coil of a Kolff twin-coil disposable artificial kidney. If the apparatus and a trained team are available, the

authors claim even better removal of salicylate than by exchange transfusion and feel that the method is indicated in all cases of salicylate poisoning showing hyperpyrexia, delirium, convulsions, coma or other signs of CNS involvement.

Other Factors

Vitamin K is needed to deal with the hypoprothrombinaemia, and vitamin C should also be given for possible capillary damage, since it has been shown experimentally that salicylates increase ascorbic acid excretion.

Care should be taken with drugs such as barbiturates, which may depress respiration, decrease CO_2 excretion and prevent the serum pH from returning to normal.

Suggested Plan of Treatment

1. Hospitalization of the patient.

2. If the patient is seen very early and is fully conscious, aspiration of stomach contents followed by lavage with water may be carried out, or alternatively an emetic may be given.

3. Half-strength Darrow's solution should be given either intravenously or by mouth, and on receipt of laboratory reports further adjustment of electrolytes may be made if necessary.

4. If the child is still acidotic after rehydration and the pH is found to be 7·15 or less, alkalis are indicated. If there are no facilities for pH studies and a considerable time has elapsed since the ingestion of the salicylate, it is probably safe to give alkalis if acidosis appears to be persisting after the child is fully hydrated. One-sixth molar lactate solution, 10 ml. per lb. body weight, given intravenously, is safer than bicarbonate, although the latter given in reasonable amounts by mouth would seem, after all, not to be harmful.

5. Vitamins K and C should be given intramuscularly.

6. In a severe case, and where facilities are available, exchange transfusion or extracorporeal dialysis may be very effective.

SUMMARY

An account is given of cases of poisoning in children admitted to 2 Cape Town hospitals over a period of several years.

Kerosene, the commonest form of poison in our series, is considered in some detail, particularly as regards the way in which it reaches the lungs.

The dangers of salicylates are stressed and current opinions are given concerning the rather complicated biochemical disturbances which occur in this type of poisoning. Controversial views on whether or not alkalis should be used in treatment are reviewed and a suggested plan of treatment is offered.

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MEAN HAEMATOLOGICAL VALUES IN ILL INFANTS AND PRE-SCHOOL CHILDREN IN CAPE TOWN*

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Previous investigations showed that iron-deficiency anaemia occurred commonly in apparently healthy infants and pre-school children in the 3 main racial groups in Cape Town. Treatment of these subjects with various oral iron preparations was followed by a significant rise in the haemoglobin level. Routine haemoglobin estimations of ill infants and children showed that they also frequently had low haemoglobin levels. In view of these findings it seemed important to determine haematological values in a series of these ill subjects.

MATERIAL

Haemoglobin estimations were done on 849 consecutive non-White infants and children attending the Out-patient Department of the Red Cross War Memorial Children's Hospital, Cape Town. Blood smears were examined and packed cell volumes and mean corpuscular haemoglobin concentration values determined in 100 of these patients whose haemoglobin levels were 0·5 g. per 100 ml. below the lowest figure accepted by Wintrobe as normal for each age group. Of the 849 patients, 501 were Cape Coloured and 348 were African. Their ages ranged from 1 week to 6 years. As relatively few White patients attend the hospital they were not included in this investigation. In a sample of 332 of these out-patiens, 49·7% of Cape Coloured and 58·7% of African infants were breast-fed up to at least the end of the third month of age.

The mean weekly income of the head of the family was £4·12 for the Cape Coloured patients and £3.16 for African patients. These figures indicate that the Cape Coloured infants and children were from the 'low income' group and the Africans from the 'medium income' group in their respective sections of the community.

The patients were taken *seriatim* from those attending the out-patient department for various ailments. They did not present primarily as cases of anaemia. The majority of illnesses were acute and usually the duration had not been more than a few days when the patient was brought to hospital. The illnesses were mainly infections, of all grades of severity. Any out-patient who showed clinical signs of dehydration or who had a blood dyscrasia was excluded. The patients investigated

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had not received iron other than that which was present in their usual diet.

TABLE I. COMPARISON OF MEAN HAEMOGLOBIN LEVELS IN ILL CAPE COLOURED AND AFRICAN INFANTS AND CHILDREN FROM 1 WEEK TO 7 YEARS OF AGE

	Cape Coloured			*African*		
Age	*No. of cases*	*S.D.*	*Mean Hb. g. %*	*No. of cases*	*S.D.*	*Mean Hb. g. %*
Months						
¼ – 1	17	3·06	14·44	2	—	14·25
1 – 2	28	2·44	11·42	13	3·93	11·35
2 – 3	28	1·23	9·88	17	0·95	10·57
3 – 4	45	1·22	9·99	31	1·40	10·37
4 – 5	35	1·43	10·19	30	1·45	10·54
5 – 6	33	1·51	9·97	12	1·02	10·38
6 – 7	33	1·61	10·27	16	1·04	9·94
7 – 8	36	1·75	10·05	27	1·47	9·97
8 – 9	25	1·55	9·79	16	1·63	9·96
9 – 10	27	2·11	9·43	11	1·27	9·74
10 – 11	22	2·13	9·47	24	2·03	9·34
11 – 12	28	1·67	9·14	14	1·88	9·82
Years						
1 – 2	49	1·98	8·81	46	1·83	9·47
2 – 3	29	1·58	9·37	25	2·09	9·42
3 – 4	11	1·44	9·99	21	1·44	11·01
4 – 5	15	1·23	10·71	18	1·00	10·82
5 – 6	20	1·96	10·66	12	0·84	11·00
6 – 7	20	1·31	10·73	13	1·24	11·39

TABLE II. COMPARISON OF MEAN HAEMOGLOBIN LEVELS IN CAPE COLOURED AND AFRICAN INFANTS AND CHILDREN FROM 1 WEEK UP TO 7 YEARS OF AGE IN APPARENT HEALTH AND ILLNESS

	Cape Coloured		*African*	
Age	*Ill Hb. g.%*	*Healthy Hb. g.%*	*Ill Hb. g.%*	*Healthy Hb. g.%*
Months				
¼ – 1	14·44	14·44	14·25	14·85
1 – 2	11·42	12·10	11·35	12·15
2 – 3	9·88	10·45	10·57	11·10
3 – 4	9·99	10·27	10·37	10·83
4 – 5	10·19	10·35	10·54	10·82
5 – 6	9·97	10·75	10·38	10·57
6 – 7	10·27	10·35	9·94	10·29
7 – 8	10·05	10·14	9·97	10·40
8 – 9	9·79	9·72	9·96	10·03
9 – 10	9·43	9·80	9·74	10·15
10 – 11	9·47	9·81	9·34	9·87
11 – 12	9·14	9·57	9·82	9·84
Years				
1 – 2	8·81	9·31	9·47	9·76
2 – 3	9·37	10·13	9·42	10·61
3 – 4	9·99	10·59	11·01	10·63
4 – 5	10·71	11·00	10·82	11·12
5 – 6	10·66	11·03	11·00	11·18
6 – 7	10·73	11·19	11·39	11·82

METHODS

Blood for haemoglobin estimation was taken by heel-prick from infants and by thumb-prick from older children, using a triangular cutting needle which gave a free, unrestricted flow without external pressure of any kind. The haemoglobin estimations were done by the oxyhaemoglobin method, using a Klett-Summerson colorimeter previously calibrated against standard haemin and cyanmethaemoglobin solutions.

Blood for packed-cell-volume determinations was taken from the internal jugular vein of infants and the anterior cubital vein of older children, and the estimations were made by the standard Wintrobe procedure.

Blood smears were stained by the May-Grunewald Giemsa method.

RESULTS

The mean haemoglobin levels in ill Cape Coloured and African infants and pre-school children are shown in Table I. Table II compares the mean haemoglobin levels in these ill Cape Coloured and African infants and pre-school children with the mean haemoglobin levels determined in apparently healthy infants and children. The mean haemoglobin values in the ill subjects were lower than the mean values in apparently healthy infants and pre-school children in Cape Town. Also, they were lower in the Cape Coloured than in the African in almost all age-period groups.

Some of the blood smears showed normal red blood corpuscle morphology while others showed the features of iron-deficiency anaemia. The lower the haemoglobin value the more likely was the smear to reveal the features of iron-deficiency anaemia.

The results of the average haemoglobin, packed cell volume and mean corpuscular haemoglobin concentration values in the 100 cases having haemoglobin levels 0·5 g. per 100 ml. below the lowest Wintrobe normal are shown in the table below:

	Mean value	*Range*	*Standard deviation*
Haemoglobin g.%	8·35	2·3 - 10·9	1·76
PCV %	31·35	8 - 45	6·56
MCHC %	26·30	17 - 31	2·79

The low mean corpuscular haemoglobin concentration in each case confirmed the presence of iron-deficiency anaemia in every infant and child in this group.

CONCLUSIONS

1. The mean haemoglobin levels in these ill Cape Coloured and African infants and pre-school children were found to be lower than the mean values in apparently healthy infants and children.

2. The mean levels in the ill Cape Coloured subjects were lower than in the Africans in almost all age-period groups.

3. The more detailed blood studies on the 100 infants and children indicated that these low haemoglobin levels were manifestations of iron-deficiency anaemia.

4. The fact that none of the cases presented symptoms of anaemia despite the low haemoglobin levels emphasizes the insidious nature of onset and slow progress of iron-deficiency anaemia in infants and children and stresses the need to administer prophylactic iron to ill infants and children.

This study was assisted by a Dr. C. L. Herman Research Grant, University of Cape Town Staff Research Fund.

FORTHCOMING INTERNATIONAL MEDICAL CONFERENCES

The Seventh International Congress of Otorhinolaryngology will be held on 23 - 29 July 1961 at the Nouvelle Faculté de Médecine de Paris. The official opening session will take place on Sunday, 23 July at 5 p.m. in the Main Amphitheatre at la Sorbonne, 47 rue des Ecoles. The scientific sessions will take place from Monday morning to Friday afternoon in the amphitheatre of the Nouvelle Faculté de Médecine, 45 rue des Saints-Pères.

The scientific programme will include 3 mornings in plenary sessions devoted to the exposition of reports and their discussion by invited guests. These sessions are: 'Present state of the pathology and treatment of otosclerosis', 'Relationship between allergy and infections of the nose and bronchi', and 'Indications and results after 5 years of surgery and radiotherapy combined with surgery in the treatment of cancers of the larynx and hypopharynx'.

For information in connection with membership of the Congress, registration fees, etc. interested doctors should communicate with Dr. H. Guillon, General Secretary, 6 Avenue Mac-Mahon, Paris 17^e, France.

South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL : VAN DIE REDAKSIE

THE MEDICAL CONGRESS

The 43rd Medical Congress of the Medical Association of South Africa will be held in Cape Town from 24 to 30 September 1961. Although it is still relatively early, the organizing committee has already started its programme of activities. The first circular letters to members (in both English and Afrikaans) were published in the issues of the *Journal* for 12 November and 3 December, in order to acquaint members in advance with the proceedings at Congress.

As was done in the past, a number of plenary sessions will be held as well as meetings of sections and groups. The Scientific Committee has decided to recommend that two plenary sessions be held and that the subjects for these sessions be *Diabetes* and *The care of the aged*. Both subjects will undoubtedly attract the attention of a great many members.

The progress which has been made in the fields of research on, and treatment in diabetes has been phenomenal. The study of diabetes has in fact become a science on its own. Moreover, it is a subject in which doctors in all branches of medicine are interested.

The problem of care of the aged is also a problem which, in recent years, has greatly increased in importance. There is a tendency, throughout the world, towards a relative and absolute increase in the number of aged persons in the community, and this gives rise to numerous new medical and social problems. The two plenary sessions should, on their own, attract a large number of doctors.

With regard to the meetings of sections and groups, the organizing committee has decided to arrange combined meetings as far as possible. This will stimulate discussion by members of related groups.

Members wishing to read papers at Congress are requested to communicate with the secretary of the section concerned as soon as possible. This will greatly facilitate the work of the Scientific Committee in arranging combined sectional meetings. The closing dates for the receipt of summaries and completed papers are 1 June and 1 July respectively. These summaries and papers should be addressed to the secretary of the section concerned through the Congress Office.

Scientific exhibitions will be organized as in the past, as well as exhibitions of medical and surgical products, and hobbies (arts and crafts). A programme of entertainment, which will include entertainment for the wives of doctors, is being planned and will include the Congress Ball and a Banquet. Honorary membership of various clubs (sporting and other) in Cape Town and its vicinity is also being arranged for members of Congress.

It is expected that one of the highlights of Congress will be closed-circuit television of surgical operations to be performed at one or more of the local teaching hospitals and projected for viewing to a full auditorium at Congress. There will be synchronous commentary and discussion by the surgeon and a panel of experts.

An intention form, which should be completed by members who intend coming to the Congress and forwarded to the Hon. Organizing Secretary, is published on page xxviii of this issue of the *Journal*. Members are requested to assist the Congress Organizing Committee by completing the intention form *now* and returning it to the Congress Office, P.O. Box 643, Cape Town.

A general Medical Congress enables members of the profession to remain abreast of progress in their own sphere of interest as well in all other fields of medicine. Members are also given the opportunity of meeting colleagues and friends with whom they may have lost touch.

In assessing the true significance of general congresses, we should, however, look beneath the surface. We should ask ourselves whether we, as a community of doctors in this country, are occupying our rightful and proper position in the medical organizations and associations of the world.

As an Association we have so far been fortunate in having been able to escape the discord and tensions that have become so prominent in other walks of public life. It is our duty, therefore, to treasure the traditional fraternity of medical men. We must build up our own intellectual and professional traditions in this country, but at the same time we must not become isolated from the great international stream of medical thought. Only by cooperating fully with other national medical associations and with the World Medical Association, will we be able to derive the greatest benefit from the greatest common source of knowledge and experience. It is in our own interest and in the interest of the patients we serve, to direct our energies towards the attainment of this goal.

VERWYDERS VAN DIE KROONSLAGARE

Die logiese grond van sekere aspekte van die behandeling van hartkramp bly selfs vandag nog ietwat duister. Daarom sal dit goed wees om sommige van die probleme in hierdie verband 'n bietjie van naderby te beskou aan die hand van 'n onlangse oorsig[1] op hierdie gebied.

Wat ons wel weet is dat die hart self, met uitsondering van die sentrale senuweestelsel, meer gevoelig is vir 'n gebrek aan suurstof as enige ander orgaan in die liggaam. Die hartspier kan nie funksioneer as daar 'n gebrek aan suurstof is nie. In hierdie opsig verskil die hartspier dus van skeletspiere, wat kan funksioneer selfs onder omstandighede van aansienlike tekort aan suurstof. Die aktiwiteit van die hart hang gedurig af van 'n genoegsame voorsiening van suurstof, wat weer gedurig afhang van die doeltreffendheid van die kroonslagaar-sirkulasie. Hartkramp ontstaan as die suurstofvoorsiening nie genoeg is vir die behoeftes van die hartspier nie.

Op die grondslag van hierdie oorwegings moet dit aan-

geneem word dat die middels wat die pyn verlig die sirkulasie in die kroonslagare verbeter. Dit moet ook aangeneem word dat, aangesien die nitriete gladde spiere laat ontspan (insluitende spiere van die slagaarstelsel), hulle (die nitriete) hartkramp verlig deur spasme van die kroonslagare op te hef. Al hierdie oorwegings is waarskynlik korrek, maar nie een van die aannames is bewys nie, aangesien geen direkte waarneming gemaak is van die kroonslagare gedurende 'n aanval, met spesiale verwysing tot die uitwerking van nitriete nie.

By miokardiale infarksie ontwikkel pyn in sy ergste vorm as gevolg van hipoksie van die miokardium. Die pyn is weerstandig vir verwyders van die kroonslagare en is moeilik om te verlig, selfs deur die gebruik van sulke pynstillende middels soos morfien.

Die middels wat gebruik word vir verwyding van die kroonslagare is betreklik non-spesifiek. Die nitriete laat alle gladde spiere ontspan, maar die arteriole is meer vatbaar vir hierdie uitwerking. Dit mag wel wees dat hulle slegs 'n betreklike spesifieke uitwerking het in gevalle van abnormale vatbaarheid, soos byvoorbeeld by spasme van die kroonslagare, waar klein dosisse verligting bring sonder dat daar 'n algemene gestelsuitwerking is. Dit veronderstel dat die middels weinig waarde mag hê vir die verbetering van kroonslagaar-sirkulasie as daar geen spasme is nie. 'n Middel wat 'n vinnige uitwerking het, is aangewese; en as die pyn eers verdwyn, skyn geen verdere medisyne nodig te wees nie. Dit mag dus gebeur dat, by die gebruik van vatverwyders wat 'n verlengde uitwerking het, die pyn reeds al deur rus verlig word voordat die farmakologiese uitwerking van die middel kan intree; ook kan die uitwerking van die middel dan nog lank voortduur nadat daar nie meer behoefte daarvoor is nie. Vir die verligting van 'n aanval van hartkramppyn is die soort middel wat 'n vinnige uitwerking het, aangewese. Daar is baie middels wat 'n stadige uitwerking het, (wat gebruik word om aanvalle te voorkom of om 'n langdurige uitwerking teweeg te bring), wat slagare wat in spasme is gou laat ontspan en die pyn dus verlig feitlik voordat dit ontwikkel. Daar is egter geen afdoende antwoord op hierdie probleme nie — in die literatuur is daar baie teensprekende verklarings oor hierdie saak. Verskeie ondersoeke op hierdie gebied is òf swak beplan òf onvoldoende gekontroleer.

Die nitriete het 'n kragtige uitwerking om die akute aanval van hartkramp te verlig. Gliserieltrinitraat (in tabletvorm) is gewoonlik die beste. Amielnitriet is nie meer amptelik (B.P.) nie, maar word tog nog aanbeveel wanneer 'n baie vinnige uitwerking verlang word. Gliserieltrinitraat wat ingesluk word (nitro-gliserien) is waardeloos, aangesien die middel in die lewer vernietig word. Die uitwerking word van krag as die middel onder die tong opgelos word; dit neem 'n rukkie langer om opgemerk te kan word, maar dit duur ook weer langer as wat die geval is met nitriete wat ingeasem word. Die middel het 'n vaste dosisvorm en word dus deur die geneesheer en nie deur die pasiënt nie, bepaal. Dit is ook veiliger om te gebruik as die geneesheer nie teenwoordig is nie. Daar is verskillende middels in hierdie ,nitraat' groep wat 'n langdurige uitwerking het, maar hulle word nie baie algemeen gebruik nie.

'n Groot groep middels wat teenhouers is van monoamien oksidase (iproniasied, isokarboksasied, fenelsien, nialamied) het nou verskyn. Hulle het 'n uitwerking op die luim van die pasiënte — die sogenoemde, psigiese energieverwekkers. Sommige hiervan mag wel van waarde wees by die behandeling van hartkramp, maar die hele aangeleentheid behoort versigtig benader te word. Aminofillien en teobromien word 'n geruime tyd lank al gebruik vir hartkramp, maar, soos dit ook die geval is met so baie ander middels, het 'n vergelyking met die gebruik van plasebo's geen spesiale voordeel aangedui nie.

Alhoewel daar dus 'n hele aantal middels is wat wel 'n uitwerking het op hartkramp, is die nitriete die enigste middels waarop ons kan reken dat hulle die kroonslagare verwyd en pyn verlig. Dit is nie bewys dat die meeste ander middels wat hierdie uitwerking sou hê, wel effektief of betroubaar is nie. Die nitriete het egter nie 'n voorbehoedende uitwerking nie, miskien omrede van die ontwikkeling van toleransie. Die groot behoefte op hierdie gebied is eintlik aan 'n middel wat aan 'n ander farmakologiese groep behoort, en waarteen toleransie nie ontwikkel nie.

1. Modell, W. (1960 - 1961): *Drugs of Choice*. St. Louis: C. V. Mosby.

DIFFUSE PLEURAL MESOTHELIOMAS IN SOUTH AFRICA

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PAUL MARCHAND, *Department of Thoracic Surgery, University of the Witwatersrand and Johannesburg General Hospital;* and
J. C. WAGNER, *Senior Medical Officer, Pathology Division, Pneumoconiosis Research Unit, South African Council for Scientific and Industrial Research, Johannesburg*

In another paper[1] we reported on 33 cases of diffuse pleural mesothelioma, histologically diagnosed at the Pneumoconiosis Research Unit of the South African Council for Scientific and Industrial Research, Johannesburg. Thirty of the cases were investigated clinically by ourselves. Since then a further 8 cases have been proved, 4 of which were under our care. This paper supplements our other communication with a description of the clinical and radiological findings in the 34 cases which we ourselves investigated.

COMPARATIVE RARITY OF DIAGNOSIS

The histological diagnosis of diffuse pleural mesotheliomas is a contentious subject. Doubt has been thrown on their existence as an entity by Robertson[2] and Willis.[3] Primary malignant mesothelial tumours were described by

Klemperer and Rabin[4] and Stout and Murray.[5] Recently reports concerning these tumours have appeared from various countries; these include those of Tobiassen,[6] Bellini and Bovo,[7] Godwin[8] and McCaughey.[9] The diagnosis in our cases has been confirmed by South African pathologists and well-known authorities from Britain and the USA, including Dible,[10] Gough,[11] Steiner,[12] Stewart,[13] and Vorwald.[14]

Diffuse pleural mesothelioma is generally considered a rare tumour. Daub and Jones[15] reported 3 cases among 345,000 consecutive admissions to the Henry Ford Hospital. The largest series that we have been able to find in the literature is that of Choptal *et al.*[16] who described 22 cases seen over a period of many years. In contrast, our larger group has been diagnosed mainly during the last 4 years. It must be stressed that we have only included cases in which the nature of the tumour has been confirmed histologically, either on biopsy or necropsy material. A number of cases showing radiological and clinical features only have been excluded. Three of these cases had markedly abnormal cells in the pleural fluid.

ASSOCIATION WITH ASBESTOS

This frequency of mesotheliomas is possibly significant in its apparent association with areas where asbestos is mined or milled. Evidence in support of this has already been presented[3] and includes:

1. The finding of asbestos bodies in the lungs of 12 (30%) of the 41 patients with mesotheliomas, even though in the majority of the earlier cases, before the association was suspected, the biopsies usually consisted of tissue from the parietal pleura only. Asbestos bodies have been found in 9 of 11 biopsies in which lung parenchyma was included. Evidence of asbestosis has been observed in 6 out of the 7 cases in which the lungs have been examined at necropsy.

2. The fact that 19 of these patients worked with asbestos, either as miners, millers or industrial workers.

3. The fact that all except 2 of the other 22 cases were either born, or spent a significant period of their lives, in the asbestos area of the north-western Cape Province.

Asbestos Bodies in the Sputum

Further, one of us (C.A.S.), working on the clinical hypothesis that exposure to asbestos causes an increased pleural reaction in patients suffering from tuberculosis, submitted numerous sputa from the West End Hospital, Kimberley, to the Pneumoconiosis Research Unit for examination. Asbestos bodies and fibres were found in the sputa of 115 patients who had no definite mining exposure. Included in these were specimens from people working in a dry-cleaning works and abattoirs in the North West Cape in places 100 miles away from the nearest mines.

The presence of asbestos bodies and fibres in the sputum is naturally only indicative of exposure to asbestos dust and is not evidence of asbestosis unless there is clinical and radiological support. However, from these findings it would appear that people possibly not associated with asbestos production or utilization, have been exposed to the hazard.

Asbestos in the North West Cape

The area of the asbestos deposit in the North West

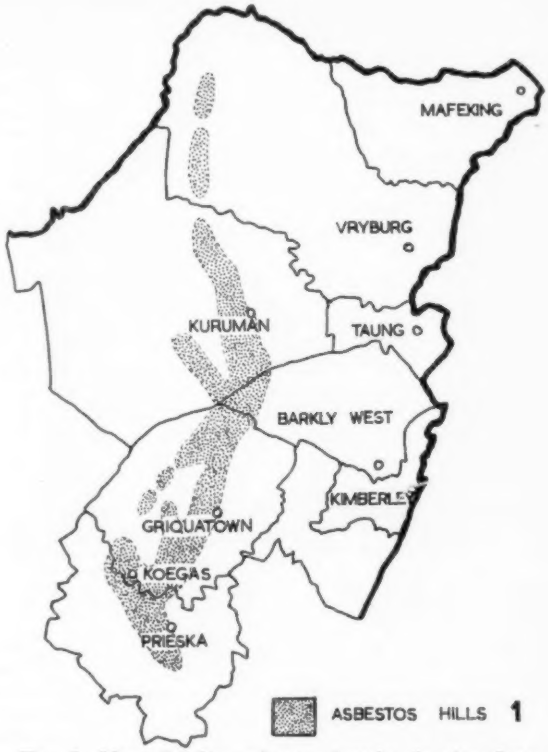

Fig. 1. Map showing asbestos deposits in the Cape Province.

Cape, the most extensive in the world, extends f[illegible] miles south of the Orange River north to the Bechuanaland border, covering approximately 8,000 square miles. The deposits are mainly in the slopes of the Asbestos Mountains, a range extending more or less longitudinally between 22·30° and 23°E (Fig. 1).

Mining and Milling

The type of asbestos mined throughout this area is crocidolite, better known as Cape blue asbestos. Crocidolite is the fibrous form of riebeckite. All stages of transformation, from massive riebeckite rock through lamellar riebeckite to asbestiform crocidolite, occur in this region. Magnetite is frequently associated with the crocidolite (Vermaas[17]). Mining of asbestos first began in the Prieska district in 1893 and gradually spread northward. In 1908, production had begun in the Kuruman district. This northward trend has continued and, in about 1950, mining started at Pom-Pom, near the Bechuanaland border. (Fig. 2 shows, graphically, the asbestos production from 1910 - 1958.) Initially the ore was quarried in numerous small open-cast workings. This was followed gradually by a type of shallow mining; inclined shafting became more common after 1930.

Since the 1939 - 1945 war, the demand for crocidolite has increased enormously. Since deeper and richer deposits have been found, vertical shafts are being sunk. However, the mines with large shallow deposits continue to use the inclined shaft and tunnel, and quarrying is still used by

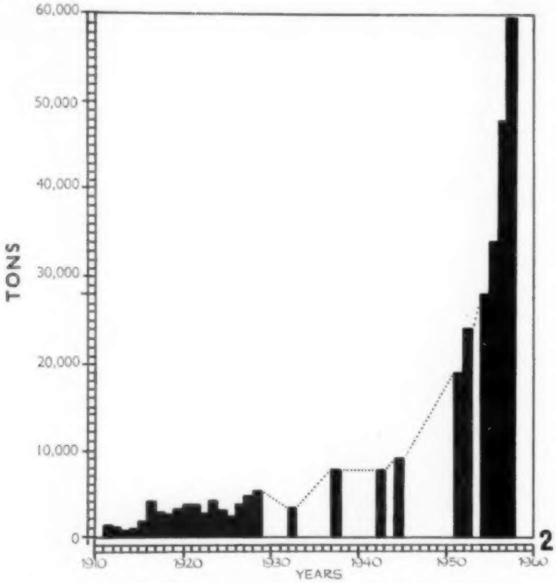

Fig. 2. Production of crocidolite by asbestos mines in the North West Cape in tons per year (1910-1958).

the few remaining smaller producers. With the building of more mills, hand cobbing* has diminished. In 1915 the first crushing mill was established at Koegas in the south. This was followed by a large mill at Kuruman (operated between 1926 and 1931) where it was situated within 300 yards of the main street, close to which hand cobbing was also done for a few years (1927 - 1930). This was followed by a mill at Prieska in 1930, which was completely re-built in 1957. Griquatown had a small mill in 1928. The practice today is for one mill to serve several mines in the immediate vicinity.

In the early days the manager and labourers lived within a few yards of their place of work, and even today the non-White personnel prefer to live as close as possible to the mills and the children play on the dumps from the mines and mills.

The industrial use of asbestos has increased during the last 30 years. In 1930 Hall stated that there were 4 firms in the Union manufacturing asbestos products (in Cape Town, Durban, Johannesburg and Meyerton) which used a total of 900 tons of fibre annually. In 1952, 17,000 tons of fibre were used in local industry. Carroll-Porczynshi[18] lists 7 firms dealing with asbestos in Cape Town and 44 in Johannesburg. Asbestos cement and an asbestos clay compound are used extensively for insulating boilers in power stations, industries and railway locomotives. Three of our patients were employed in this way. Another patient was employed manufacturing fire-proof clothing. Cases of asbestosis have been observed in a variety of industries on the Witwatersrand.

Asbestos and Carcinoma

The association of asbestos with carcinoma of the lung was first described by Lynch and Smith in 1935.[19] By 1955, according to Doll,[20] a total of 61 cases had been reported. Merewether[21] analysed the cases that had occurred in Britain between 1924 and 1954. He found that in a total number of 344 cases, carcinoma of the lung was present in 55 (16%), the figure for males being 41 (20% of males) and for females 14 (10% of females). Six cases of mesothelioma of the pleura associated with asbestosis have been described. One was mentioned by Doll,[20] 2 by Cartier,[22] and 3 by Van der Schoot.[23] A further 33 cases with possible asbestos exposure were described by Wagner *et al.*[1]

In our series of cases the latent period, between first apparent exposure to asbestos dust and the initial symptoms of the disease, was between 20 and 60 years. In some cases the patients had left the North West Cape and, in others, the exposure had been of such a transitory nature that it was rapidly forgotten. Evidence of exposure was only obtained after long and detailed questioning. According to Day[24] and Stewart,[13] a long latent period between initial exposure and the development of evidence of malignancy is one of the features of occupational tumours. The reason for the tumours having been found in the vicinity of the Cape asbestos fields and not in the region of the Transvaal asbestos mines may be due to this time factor, assuming that the association is significant, since the industry was established earlier in the Cape than in the Transvaal, where large-scale mining began comparatively recently. The type of asbestos mined is another possible factor which would apply to the chrysotile mines of the Eastern Transvaal. However, the amosite fibre found in the Lydenburg district is similar to the Cape crocidolite and the crocidolite mined around the Pietersburg district is identical.

CLINICAL PICTURE OF MESOTHELIOMA

Clinical Presentation

The majority of patients are over the age of 40, and usually present with symptoms suggestive of a primary tuberculous effusion. Pain varies from a feeling of heaviness to acute pleuritic pain. From the beginning or after a variable time there may be increasing dyspnoea on exertion. Cough is not a predominant symptom in this group. A few patients present as cases of acute respiratory infection with a productive cough. This is accompanied by a pleural effusion. In some cases there is an interval between the respiratory infection and the finding of the pleural effusion. The course of the disease, while ultimately similar, usually takes one of two forms — either the pleural effusion continues to increase, requiring frequent paracentesis, or it undergoes a temporary quiescent period, while the pleural space becomes obliterated by tumour. Towards the end the patient is seriously disabled through pleuritic pain and dyspnoea. Terminally ascites may develop due either to peritoneal metastases or to cardiac failure.

Clinically the disease may be described as having 3 stages. Initially the presentation is one of pleurisy and bronchitis and the usual diagnosis is a virus or pyogenic pneumonitis. At this stage antibiotics are usually successful in alleviating the symptoms. The second stage is one of

* Freeing the fibre from the ore by striking the lumps of rock with a hammer.

pleural effusion with pleural thickening; it is then that the patient is usually admitted to hospital for investigation. Tuberculosis, malignancy, asbestosis and other causes of pleural disease may be considered. The final stage is one of obvious thoracic malignancy.

Age, Sex, Race, Habitat and Occupation

Our 34 cases consisted of 24 males and 10 females (Table I). Six men who had lived all their lives in the vicinity of the asbestos mines were the only patients under 40 at the time of diagnosis. Four of them had been miners. They were all in their thirties, the youngest being a miner aged 31. On the other hand, 7 patients were over 60, 5 being men and 2 women. The oldest was a man of 68. Six of these patients had lived for many years in the asbestos area and 3 of the 5 men had worked for long periods in close contact with asbestos. One woman died at the age of 60. As far as we have been able to ascertain, she had never been in contact with asbestos in her occupation as a nursing sister, and had never lived in the North West Cape. She is the only patient of the 34 who apparently had no contact whatever with asbestos.

TABLE I. AGE AND RACE DISTRIBUTION OF THE 34 CASES OF MESOTHELIOMA DESCRIBED

Race	Sex	Age 30 - 39	40 - 49	50 - 59	Over 60	Total
White	Male (14)	2	6	3	3	20
	Female (6)	0	1	3	2	
Coloured	Male (5)	2	2	0	1	8
	Female (3)	0	2	1	0	
Bantu	Male (5)	2	2	0	1	6
	Female (1)	0	0	1	0	
Total		6	13	8	7	34

The remaining 21 patients, 8 women and 13 men, were aged between 40 and 60. The 8 women had either been born in the asbestos district, or had moved there in early childhood. At the time of diagnosis, 3 of these women lived in the Transvaal and had never, or only occasionally, visited the North West Cape after leaving school. Three women had been engaged in the cobbing of asbestos in their homes. The non-European women mostly lived close to mines in houses where cobbing operations produced much dust, even when they themselves had done no such work. The European women had been to school near mills or had played on the dumps.

The 13 men aged between 40 and 60 comprised 7 whose work was connected with the mining, milling and transport of asbestos, 2 who were born and bred in the district but had not been engaged in the industry (they had often watched cobbing operations in native huts and had played on dumps and in mine shafts on farms), and 4 who worked with asbestos in distant industrial towns. Of these industrial cases, 2 were boiler makers who had lagged locomotive boilers. Another of these men had spent the war years manufacturing asbestos protective devices for fighting aircraft fires, and the fourth had worked as a boiler maker in a Cape factory, whose supply of asbestos was obtained from the Cape asbestos fields. It is interesting to note that in 1 family both the father and a daughter died of histologically proved mesotheliomas. In 2 other cases a sibling had radiological evidence of asbestosis.

Physical Signs

In the early stages auscultatory evidence of pleurisy, bronchitis, or consolidation are usual. As the disease progresses, respiratory excursions diminish until eventually a frozen chest results, with flattening of the infraclavicular region, 'roof-tiling' of the ribs and immobility of the chest wall. The chest is then stony-dull to percussion and air entry is grossly impaired.

During this late stage of the disease, evidence of secondary spread may appear. Eight cases in our series showed obvious clinical signs of peritoneal involvement before death. The peritoneum is usually infiltrated by direct spread through the diaphragm. The liver may be enlarged and nodular, due to perihepatic involvement. Localized masses due to involvement of the omentum or para-aortic lymph nodes may be felt. In these cases ascites is common. Involvement of the lymph glands of the neck has been clinically obvious in 2 cases. Implantation of the tumour in a biopsy scar is not frequent, but it complicated 4 thoracotomies and 1 thoracoscopy in our series. Haematogenous spread is unusual and only 2 cases have eventually developed distant metastases. These were predominantly subcutaneous but, in 1 instance, numerous small deposits appeared in the tongue.

Pleural effusions have always developed. Fluid collections of 5 - 6 pints have occurred, but often the pleural space is largely obliterated and only small effusions can collect. On tapping these effusions, considerable resistance is encountered due to the thickness of the diseased tissue (up to 2 cm.) which has to be traversed. Even with the point inside the cavity, the needle is often so tightly gripped that manipulations are restricted. Usually the fluid is straw-coloured, but blood-staining occurs and may be heavy.

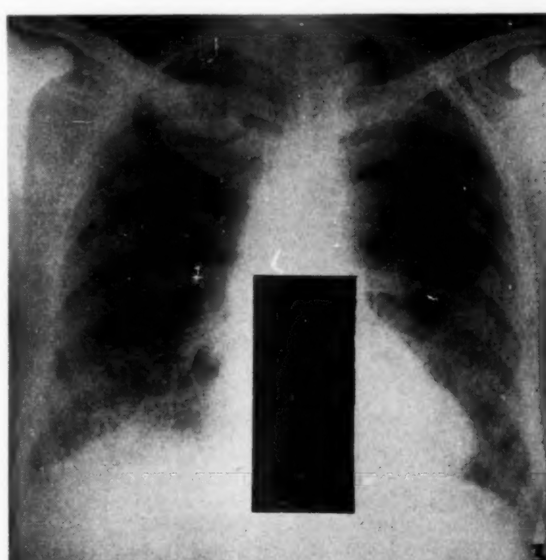

Fig. 3. **Diffuse pulmonary asbestosis, particulary well shown in lower lung zones.**

SPECIAL INVESTIGATIONS

Radiological Features

The X-ray findings roughly parallel the clinical progress. The initial radiographs may show few changes. In about a third of the cases, radiological signs of asbestosis were present. Asbestosis, in individuals with a proved and adequate record of exposure, is characterized by evidence of diffuse interstitial fibrosis of varying degree. In a well-established case the appearance will conform to the well-known classical pattern of pulmonary asbestosis. This may be either a generalized homogenous clouding of the lung fields, particularly the lower zones, or a fine striate and fibrillary change in the lung structure, with progressive loss of pulmonary radiolucency (Fig. 3).

In the majority of cases of asbestosis, old-standing pleuritic changes are also seen. Thus a combination of both pleural and pulmonary parenchymal pathology frequently occurs. The pleural changes take the form of bilateral pleural thickening, pleural adhesions and rather characteristic dense calcific plaques (Frost *et al.*[25]). While pleural thickening *per se* in asbestosis is non-specific in character, sclerotic pleurisy, with plaque formation, constitutes a readily recognizable and rather typical entity (Fig. 4). The pattern of calcification patently differs from

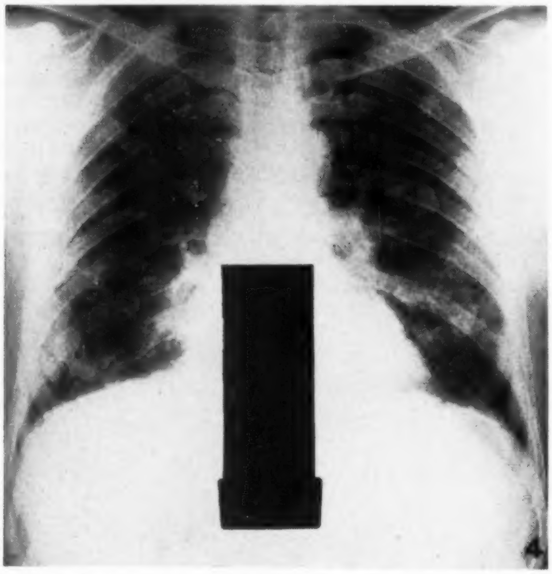

Fig. 4. Extensive bilateral pleural plaque formation. Typical involvement of paramediastinal and diaphragmatic pleura is also demonstrated.

pleural calcification due to other causes. This pattern was recognized frequently during the recent investigations of miners and millers. Even the chance finding of such typical calcification is now accepted as being highly significant, so that previous exposure to asbestos may be predicted with reasonable confidence. The dense plaques, however, do simulate those described in workers exposed to other silicate dusts such as tremolite talc, calcimine and mica (Smith[26]). The plaques may be few or widespread. They are usually bilateral, and disposed in irregular patches, chiefly in the middle and lower zones. Seen end-on they appear as linear plaques in the periphery, along the diaphragmatic contours, and adjacent to the mediastinum. A lateral view of the thorax will also very often show extensive linear plaque formation, involving the diaphragm and the anterior aspect directly behind the sternum. Such pleural calcification was encountered in several of our cases of mesothelioma (Hurwitz[27, 28]).

However, in the majority of cases, no evidence of preceding pleural or pulmonary asbestosis was found. These cases, on investigation, invariably showed unilateral pleural involvement in the form of diffuse thickening or effusion. The appearance may be massive from the beginning, but usually a localized scalloping, or solitary mass in the periphery, raises the first suspicion of pleural neoplasm. As the disease advances, more extensive nodular or 'lumpy' pleural thickening develops (Fig. 5). This pattern

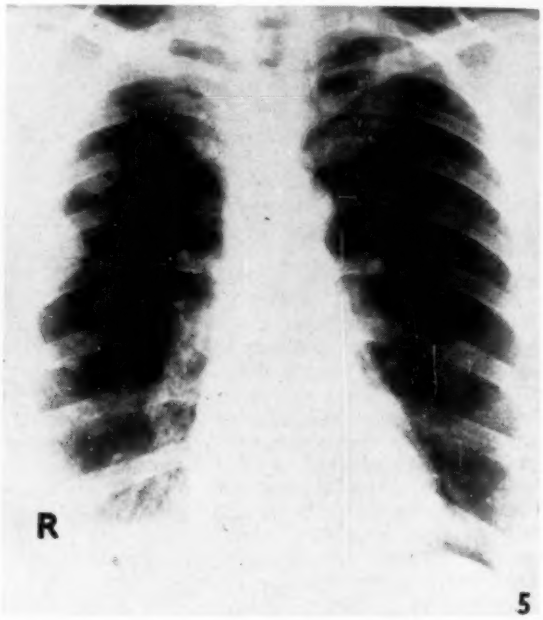

Fig. 5. Nodular pleural thickening, seen clearly on the right.

of unilateral pleural pathology is a highly significant finding in a patient from the asbestos areas, but of course, a similar appearance may be produced by secondary malignant involvement of the pleura. Very often a large pleural effusion will obliterate the picture and, only after removal of the fluid, will it become apparent that the pleura is grossly thickened and nodular. Both the parietal and visceral pleura are affected and an induced pneumothorax, particularly, will clearly demonstrate the rather characteristic pattern of marginal massive nodularity along the parietal chest wall, over the surface of the collapsed lung, and at the base (Fig. 6).

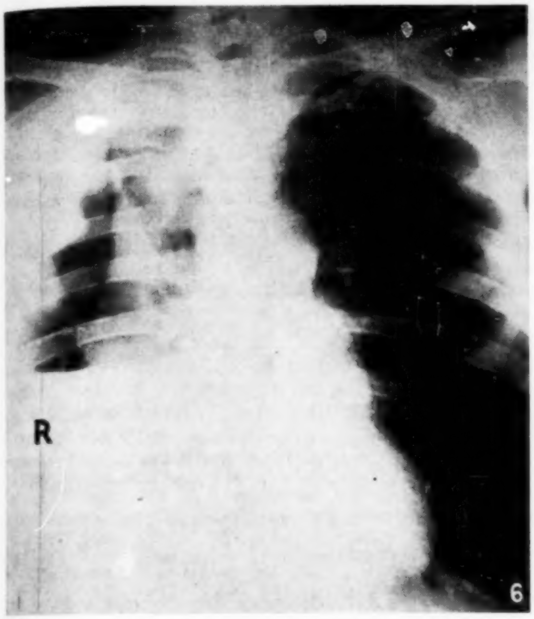

Fig. 6. Induced pneumothorax revealing gross malignant involvement of pleura. A considerable quantity of fluid is still present in the right pleural cavity.

In the later stages, a considerable part of the pleural cavity may be obliterated and a comparatively small space may remain. Here fluid may collect under tension and the lung may be further compressed, in spite of being encased by a thick tough covering of malignant tissue. As the mediastinal pleura thickens, and the regional lymph glands become infiltrated, widening of the mediastinum is seen radiologically. Eventually the pericardium is involved by direct extension, and the resultant pericardial effusion causes progressive enlargement of the cardiac silhouette and a change in its configuration. Extrapleural extension can also occur; then radiological evidence of rib involvement may be present.

Stout and Murray[5] remark upon the right-sided preponderance of the disease. In the series of 24 cases reported by Choptal *et al.*,[16] 19 were right-sided. Our cases present an identical distribution, since 27 of the 34 cases had right-sided lesions. In no case was the mesotheliomatous process initially bilateral, although the X-rays of 10 of the patients showed bilateral changes due to asbestosis.

Thoracoscopy and Thoracotomy

The information obtained from thoracoscopy was disappointing. The view is always limited in cases of mesothelioma because the cannula cannot be freely manipulated. When blood has extravasated into the effusion, the walls of the cavity are covered with fibrinous deposits and no architectural details are visible. The chief objection to the technique, as a diagnostic measure, is that the choice of area for biopsy is restricted, and it is impossible to obtain a representative specimen. Thoracoscopy was the usual diagnostic procedure in the early stages and, although sufficient material for microscopic examination was usually obtained, we feel that a great deal of information was missed.

With early cases, where the pleura is thin and the effusion clear, it is often possible to visualize the discrete pleural nodules overlying the still elastic lung. The nodules are then usually cherry-coloured and either smoothly-rounded or warty in appearance. Later the discrete nodules coalesce and the underlying lung is obscured. The growth is then usually seen as a grey opaque membrane, with localized excrescences of variable size and similar appearance projecting into the pleural space. At this stage the outstanding feature is the thickness and density of the pleural growth and the complete immobility of the incarcerated lung beneath.

As far as possible, it is now our policy to obtain a specimen of lung by open operation. With 4 early cases, attempts were made to decorticate lungs which were completely encased by thick, apparently fibrous, coverings. In 3 cases the findings were similar. The parietal pleura was 1 - 2 cm. thick and very hard. The lungs were collapsed and bound down by thickened visceral pleura, identical in appearance with the parietal pleura. The pleural spaces were occupied by straw-coloured or blood-stained fluid. Attempts to strip the 'peel' off the chest wall could only be made through the extrapleural plane, but even then the intercostal spaces were constantly entered and no vestige of normal parietal pleura could be found. Calcified plaques within the diseased tissue were frequently encountered, particularly over the diaphragm and inter-

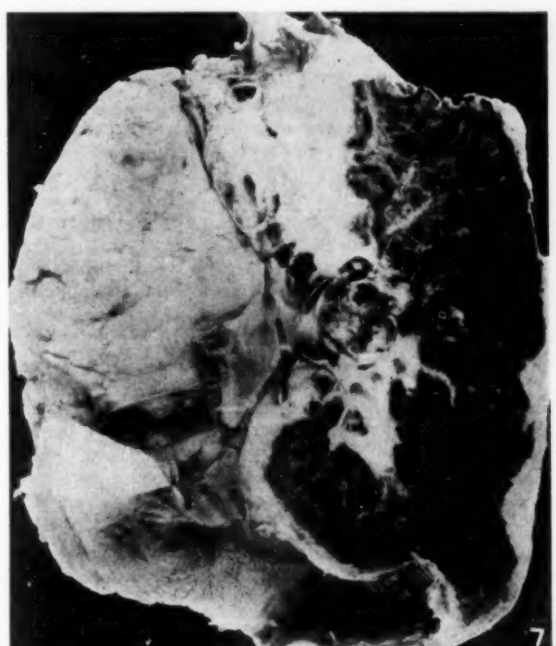

Fig. 7. Macroscopic specimen of mesothelioma of the pleura.

lobar fissures. It was never possible to define planes between the visceral pleura and the pathological tissue, so that separation was possible only by entering lung tissue. False planes could be developed through compressed peripheral lung. When the deep aspects of the peel were inspected, fine blackened fragments of lung were seen adhering to them. The tumours dipped into the lung substance along the interlobular septa and, for this reason, stripping along these false planes was halted every few millimetres. Inevitably, the procedure has to be abandoned, leaving a collapsed, tattered and leaking lung.

AUTOPSY FINDINGS

At autopsy the whole thoracic cavity may be occupied by a large gelatinous tumour, which displaces the mediastinum, and markedly compresses the lung (Fig. 7). In other cases the tumour may be dense and cartilaginous. In these advanced cases no remnant of a pleural space may remain. The tumour infiltrates adjacent structures, so that the pericardium, heart, chest wall, diaphragm and liver may form one solid mass. In 1 case the tumour extended into the posterior peritoneum, encasing a kidney. The mediastinal, cervical and para-aortic lymph glands may be involved and appear as large discrete tumours. The lung is always compressed, with marked lymphatic spread in some terminal cases. Because the neoplasm extends along the interlobar fissures, the lobes appear as 2 or 3 small deeply pigmented areas within a huge pinkish-white mass.

PROGNOSIS AND TREATMENT

Our first impressions, based upon the apparently fibrous nature of the tumour, was that it was slow-growing, but we found this to be far from true. According to Choptal *et al.*[16] the average duration of life from the time of onset of symptoms is 18 months. Hochberg's review[29] of 43 cases showed that 37 (86%) died within the first year. Of the 34 patients discussed here, 28 have died, 17 (61%) within a year of the development of symptoms. Seven (25%) died during the second year, 2 (7%) during the third year, and all 28 were dead within 48 months, which is the longest survival time among our patients.

It has already been shown that operative removal of the growth is impossible and some form of chemo- or radio-active therapy would seem to provide the only hope of effective treatment. Results have been disappointing. Two patients were given repeated intrapleural injections of nitrogen mustard. Both men died within 6 months. Four patients, with seemingly localized areas of disease, were given deep-X-ray therapy. All were dead within a year. Six patients have had radio-active gold instillations. Four died within 13 months of the commencement of their injections and 2 are alive, but their treatment was recent. There seems little to be gained by subjecting patients to the additional discomforts of present forms of therapy, but we still advise radio-active gold intrapleurally for the comparatively early case, where the lung retains some elasticity.

SUMMARY

The clinical and radiological findings of 34 patients suffering from diffuse pleural mesotheliomas are described. Thirty-three of these patients gave a history of exposure to crocidolite asbestos mined in the North West Cape. However, even when lung tissue was available, histological evidence of asbestosis was not obtained in every case. Asbestos bodies were found in the lung tissue of 30% of the cases.

The findings of this and our other paper[1] are sufficiently striking to justify further epidemiological and laboratory investigations. These investigations will attempt to determine the causative and correlative factors, including a possible relationship with asbestos or other elements, especially in the regions from which the cases have most frequently been reported.

We wish to thank the many medical practitioners in the Griqualand West District, and the thoracic surgeons, physicians, radiologists and pathologists from elsewhere in the Union whose assistance has made this investigation possible.

We thank the Director of the Pneumoconiosis Bureau for permission to reproduce the X-ray plates shown in Figs. 3 and 4.

We thank the Secretary for Health for permission to publish this paper.

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PASSING EVENTS : IN DIE VERBYGAAN

South African Institute for Medical Research, Johannesburg. Staff Scientific Meeting. The next meeting will be held on Monday 23 January at 5.10 p.m. in the Institute Lecture Theatre. Dr. L. Webster, of the Pneumoconiosis Research Unit, will speak on 'Pneumoconiosis problems'.

* * *

Southern Transvaal Branch (M.A.S.A.). An important general meeting of this Branch will be held at Medical House, 5 Esselen Street, Hospital Hill, Johannesburg, on Tuesday 17 January 1961 at 8.15 p.m. Reports will be presented by the President on the recent developments in connection with (*a*) medical insurance companies, (*b*) medical aid societies, and (*c*) the Medical Services Plan. The Domicile Clause in Section 24 of the Medical, Dental and Pharmacy Act will also be before the meeting for full discussion. Members of the Branch are urgently requested to attend this important meeting.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

SOUTH AFRICAN MUTUAL MEDICAL AID SOCIETY

It is with regret that members of the Association are informed that negotiations with the South African Mutual Medical Aid Society have broken down, as a result of which approval cannot be granted to the Society. This means that *all persons insured by the Society,* including the groups already approved (p. 1064 of the *Journal* for 10 December 1960), must now be treated as private patients to whom accounts should be rendered direct and who should be personally responsible for the payment of their accounts.

Cheques forwarded to practitioners by the South African Mutual Medical Aid Society for a sum less than that of the account rendered to the patient should be returned to the insurance company or the patient forthwith.

SUID-AFRIKAANSE ONDERLINGE MEDIESE HULPVERENIGING

Dit is met spyt dat aangekondig moet word dat onderhandelinge met die Suid-Afrikaanse Onderlinge Mediese Hulpvereniging afgebreek is, as 'n gevolg waarvan dié Hulpvereniging nie goedgekeur kan word nie. Dit beteken dât *alle persone wat deur die Hulpvereniging verseker is,* insluitende die groepe wat reeds goedgekeur is (p. 1064 van die *Tydskrif* van 10 Desember 1960) nou as private pasiënte behandel moet word aan wie rekenings direk gestuur moet word en wat persoonlik vir betaling van hul rekenings verantwoordelik is.

Tjeks wat deur die Suid-Afrikaanse Onderlinge Mediese Hulpvereniging aan geneeshere gestuur word, wat uitgemaak is vir 'n bedrag minder as die bedrag van die rekening wat aan die pasiënt gelewer is, moet sonder meer aan die Hulpvereniging of aan die pasiënt teruggestuur word.

EXPLANATORY STATEMENT : SOUTH AFRICAN MUTUAL MEDICAL AID SOCIETY

When the insurance companies entered the field of medical insurance they created their own tariffs which amounted to the medical aid rates plus a percentage (between 25 and 33⅓%). They fixed premiums at a level which the Association always considered too low for the benefits they offered, but this was all done without contact with the Medical Association. Later they approached the Association and arranged that we should assist them and provide a fixed tariff.

The Federal Council of the Association, at its Meeting held in March 1960, agreed to grant the preferential tariff for medical aid societies to the members of groups administered by insurance companies which conformed with the rules laid down for approval as medical aid societies. This resolution of Council followed on thorough discussions with the insurance companies and was welcomed by them as a step forward in providing assistance to members of the public who fell within the medical aid category. In addition Federal Council was satisfied that the medical aid system was being extended to those persons who were eligible for such consideration. The fundamental principle for medical aid societies was the provision of a preferential tariff of fees for people in the middle and the lower-middle income groups in return for guaranteed payment of the full fee by the medical aid society.

At its Meeting held in October 1960, the Federal Council agreed, after having received representations from the insurance companies, to amend the rule regarding the income of members of medical aid societies. Whereas members of employer-sponsored societies would continue to comply with an average income figure, the groups underwritten by insurance companies would comply with an income ceiling. This meant that all insured persons with an income below the ceiling would be regarded as medical aid patients on condition that the insurance companies observed all the other conditions in the rules to which medical aid societies have to conform.

Following this Federal Council Meeting further discussions were held with the insurance companies and finally a meeting took place in Pretoria on 6 December between the Executive Committee of Federal Council and the representatives of the insurance companies. At this meeting it was agreed that the income ceiling be fixed at £2.300 *per annum* and all insured persons below this income would qualify for the group to receive the preferential tariff for medical aid societies. A satisfactory agreement was reached with the representative of SANSOM regarding the recognition of a group whose members would receive the preferential tariff. The representative of the South African Mutual Medical Aid Society was unfortunately not in a position to make any alterations in the rules of the scheme submitted or to bind his Society by any decision.

The South African Mutual Medical Aid Society submitted two schemes 'A' and 'B', of which scheme 'B' was approved. The new scheme 'A', which the Society had already established, did not conform with the rules for medical aid societies, in that, *inter alia*, it made the doctor entirely responsible for collecting the fees for the first two visits or consultations in every month, and placed undue limitations on the total number of visits or consultations for which the Society accepted responsibility as well as on the percentage benefits to which members would be entitled. The acceptance of this would have resulted in the undermining of the agreements reached over the years with employer-sponsored societies. The granting of recognition and the offer of the preferential tariff was contingent on the acceptance by the insurance companies of the rules and regulations which applied to medical aid societies, and this was rejected. Furthermore, the Association was informed that it was the intention of the Society to divert as quickly as possible all the members of Scheme 'B' and those of groups already approved to Scheme 'A'. Meanwhile, the Society was unable to submit an acceptable method of distinguishing between the members of Scheme 'A' and Scheme 'B' for the benefit of the profession.

Certain coded cheques issued by the South African Mutual Medical Aid Society had caused considerable confusion and dissatisfaction in the ranks of the profession. These cheques, made out for an amount less than the account rendered to the patient, were tendered 'in full settlement' of the doctor's account and acceptance of the cheque precluded the doctor from collecting the balance of the account from the patient. The Society had previously been informed of the disturbance which these cheques had caused and had agreed not to issue any further cheques while negotiations were in progress. The Society now insisted, however, that despite the non-approval of Scheme 'A' it would continue to issue coded cheques in respect of medical services rendered to the members of that scheme.

Federal Council, when it discussed these coded cheques at its Meeting held in October 1960, passed the following resolution: 'That no further negotiations regarding medical aid groups will be undertaken with insurance companies, and no further groups will be recognized, until satisfactory arrangements re matters affecting the dignity of the profession and payment of fees have been finally settled between the Medical Association and the insurance companies.'

In consequence of the above, there was no other course open to the Association but to terminate all further negotiations. The Executive Committee of Federal Council therefore resolved to break off all further negotiations with the South African Mutual Medical Aid Society and to notify members of the Association accordingly. This means that *no groups of persons insured by the South African Mutual Medical Aid Society*, including the groups approved by Federal Council in October 1960 (see p. 1064 of the *Journal* for 10 December 1960), will be recognized and that members of the Association are advised to return to the Society or to the patient all cheques made out for an amount less than that of the account rendered to the patient.

L. M. Marchand
Associate Secretary

28 Plaza Building
Pretoria
5 January 1961

From the Secretary's Desk

Medical Insurance

Elsewhere you have been informed officially that the negotiations between the Association and the South African Mutual Medical Aid Society have broken down and that in future *all* members of the Society should be treated as ordinary patients irrespective of the fact that they have sought to insure themselves and their families against sickness.

The negotiations have been protracted and the Association has gone a long way to meet the Society in order to make recognition and approval more possible of attainment; but, as the Chairman of the Board of the South African Mutual Life Assurance Society reported at the Annual General Meeting, the Society's losses have been fairly considerable. Naturally it was necessary to change the scheme, and the changes have been so drastic that the Association could not approve of the scheme now being offered, although the former scheme was acceptable.

The main cause of the breakdown has been the persistence of the Society in sending to doctors cheques which were based on the tariff of fees for *approved* medical aid societies with the insistence that they be accepted 'in full settlement'. This attitude has been resented by most doctors as high-handed and as an interference in the normally cordial relationship which exists between doctor and patient. The fact that some of the cheques sent to doctors are reported to have been less than the tariff charges (presumably due to 'clerical errors') has not helped to improve the position.

You will appreciate that I have had many complaints brought to my notice and they have not been from doctors only. Many of the patients who have complained to me have appreciated the doctors' point of view when the position has been explained to them. The fact that the Society has refused to discontinue sending its 'coded' cheques to doctors is the direct cause of the breakdown, as it was resolved at the last Meeting of the Federal Council 'that no further negotiations regarding medical aid groups will be undertaken with insurance companies and no further groups will be recognized until satisfactory arrangements re matters affecting the dignity of the profession and payment of fees have been finally settled between the Medical Association and the insurance companies'.

One of the main objects for which the Association was established is to 'maintain the honour and interests of the medical profession', and we would be failing in our duty to our members and our colleagues, as well as to our patients, if we allowed ourselves to be dictated to by any business concern in any way whatsoever.

It would be as well to look back and to realize that the South African Mutual Medical Aid Society commenced operations without any reference to the Association and originally used the tariff book as a guide to pay fees which were 25% higher than the tariff against subscriptions which were obviously too low for the services offered. We know that originally some doctors asked the Society to pay them direct and apparently the Society was only too willing to do so. We know now that the desire to do so is based on a policy of providing 'disincentives', for no matter what the doctor charges there will always be an amount which will be owed to the Society by its member, whereas if the Society paid its member direct what is due to him by way of indemnity it is possible, and in some cases probable, that the doctor's charges would be less than the indemnity paid.

In endeavouring to cooperate with the South African Mutual Medical Aid Society, the Association was willing to recognize all members of the original schemes earning up to the comparatively high ceiling income of £2,300 *per annum*, provided that only for these recognized persons would the Society pay the doctor direct and in full according to the tariff. For all members of their new schemes of which the Association cannot approve and for those with incomes over the £2,300 ceiling, the Society was to pay the patient, from whom the doctor would collect his fees as is normal custom. The Society, however, wishes to continue to pay doctors with 'coded' cheques for members of its unapproved schemes as well, presumably for reasons of 'disincentive', in spite of the fact that this is considered to be derogatory to the dignity of the profession and is certainly opposed to both its honour and its interests.

The matter would seem to be becoming a trial of strength between the Association and 'big business'. Fortunately we are a voluntary organization composed of professional men and women who *want* to belong to their own Association and who want to see it strong and undivided. This, then, is our strength, and by standing together once again we shall continue to maintain our honour and interests as we did in the struggle with the Transvaal Provincial Administration in the middle forties.

Following the Official Announcement, it behoves all members to inform all their patients that members of the South African Mutual Medical Aid Society (including those groups mentioned in the *Journal* of 10 December 1960, from which approval has now been withdrawn) that they will be treated as ordinary patients from whom fees will be claimed and that they must make their own arrangements with the Society in regard to indemnity. This may be done directly by the doctor or through his receptionist, or, in the case of general practitioners, to all *bona fide* patients by means of a personal letter pointing out that this is done at the request of the Medical Association of South Africa. It would be as well also to write to the Manager of the South African Mutual Medical Aid Society whose address is P.O. Box 90, Howard Place, Cape, informing him that you are not prepared to accept any more cheques from the Society and will look to your patient for settlement.

I need hardly stress the importance of a firm stand by all practitioners. Once again we have the opportunity of showing that we are a united profession who cherish our freedom to practise our art and science for such recompense as we may claim according to our own consciences and not according to the dictates of any business house, and in this we know we can depend on the loyalty and oneness of our colleagues.

Medical House
Cape Town

MEDICAL ASSOCIATION OF SOUTH AFRICA : MEDIESE VERENIGING VAN SUID-AFRIKA

MINUTES OF THE ANNUAL GENERAL MEETING OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA, HELD IN THE UNION STEEL CORPORATION RECREATION CLUB, VEREENIGING, ON WEDNESDAY, 19 OCTOBER 1960, AT 9.30 A.M.

Present: The President (Dr. P. F. H. Wagner) and 60 members.

The President welcomed the members to the meeting.

1. *Notice convening the meeting*, which had been published in the *Journal* of 3 September 1960, was taken as read.

2. *Minutes of the Annual General Meeting and Adjourned Annual General Meeting* held in East London on 24 and 28 September 1959, respectively, which had been published in the *Journal* of 30 January 1960, were taken as read and *Confirmed*. They were signed by the President.

3. *Annual Report of Chairman of Council*, published in

the *Journal* of 6 August 1960: It was proposed by the Chairman of Council, Dr. J. H. Struthers, and *Resolved* that his Report be taken as read and adopted.

4. *Annual Report and Balance Sheet for the year ended 31 December 1959*, published in the *Journal* of 30 July 1960: It was proposed by Dr. Struthers, seconded by Dr. Wagner and *Resolved* that this be adopted.

5. *Election of auditors:* It was proposed by Mr. J. D. Joubert, seconded by Dr. Wagner and *Resolved* that Messrs. Gurney, Notcutt & Fisher be reappointed as the Association's auditors for the year 1961, at the remuneration of £250 *per annum*.

6. *Induction of President:* The retiring President, Dr. Wagner, expressed his appreciation of the honour which had been bestowed on him in his election as President of the Association for the past year.

Dr. Wagner then inducted the incoming President, Dr. W. Chapman, and, in doing so, wished him a pleasant and successful year of office. Acclamation.

Dr. Chapman took the Chair and thanked the members for the high honour bestowed on him in his election as President. He gave his assurance that he would always do his best in the interests of the profession.

On behalf of the Vaal River Branch he welcomed the members of Federal Council to Vereeniging and wished them a pleasant stay in the city. He expressed the hope that the Council's deliberations would redound to the honour and dignity of the profession. Acclamation.

Dr. Struthers proposed a vote of thanks to Dr. Wagner for the able manner in which he had carried out his duties as President. He mentioned that Dr. Wagner had been the Association's official delegate at the recent Annual Meeting of the British Medical Association, and stated that although Dr. Wagner had not always been in the best of health he had attended most assiduously to the affairs connected with his office. On behalf of the Association he wished Dr. Wagner well in the future. Acclamation.

Dr. Chapman then declared the meeting to be adjourned until 8 o'clock that evening.

ADJOURNED ANNUAL GENERAL MEETING OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA HELD IN THE UNION STEEL CORPORATION RECREATION CLUB, VEREENIGING, ON WEDNESDAY, 19 OCTOBER 1960, AT 8.30 P.M.

Present: The President (Dr. W. Chapman), the Immediate Past President (Dr. P. F. H. Wagner), the Mayor of Vereeniging, the Chairman of Federal Council (Dr. E. W. Turton), the Immediate Past Chairman of Council (Dr. J. H. Struthers), members of Federal Council and the Vaal River Branch, and their ladies. The Secretary of the Association (Dr. A. H. Tonkin) was in attendance, and certain other guests were also present.

The meeting opened with prayer led by the President's chaplain.

The Mayor of Vereeniging welcomed the delegates to the city. He remarked that this was the second occasion on which a meeting of Federal Council was being held in Vereeniging, and he expressed the hope that the deliberations and decisions of the Council would be to the benefit of both the medical profession and the public.

He congratulated Dr. Chapman on the honour which his colleagues had bestowed on him by electing him as President of the Association, and he wished him a successful term of office. Acclamation.

Award of Association's Bronze Medal to Dr. W. Chapman and Dr. C. Adler: The Secretary read citations concerning the recipients, stating that the awards were made in recognition of meritorious service to the Association.

The Medals were presented to Dr. Chapman and Dr. Adler amid acclamation.

Presentation of Insignia: Dr. Chapman presented to Dr. Wagner the insignia of Immediate Past President, Mrs. Wagner presented to Mrs. Chapman the badge of office of President's Lady, and Dr. Chapman presented to Dr. Struthers the insignia of Immediate Past Chairman of Federal Council. Acclamation.

Presidential Address: Dr. Chapman then delivered his Presidential Address on '50 years in Vereeniging'. His Address was received with acclamation.

The meeting ended at 9.30 p.m. and was followed by a reception at which the members and guests were received by the President and Mrs. Chapman.

SOUTH AFRICAN MEDICAL AND DENTAL COUNCIL : SUID-AFRIKAANSE GENEESKUNDIGE EN TANDHEELKUNDIGE RAAD

RULES REGARDING THE REGISTRATION OF ADDITIONAL QUALIFICATIONS

The Minister of Health, in exercise of the powers conferred on him by sub-section (4) of section *ninety-four* of the Medical, Dental and Pharmacy Act, 1928 (Act No. 13 of 1928), has approved the following amendment of the rules made by the South African Medical and Dental Council under paragraph (*h*) of sub-section (2) of the said section of the Act and published under Government Notice No. 2440 of the 15th December, 1955, as amended:

(i) By the deletion of the following qualifications under the heading:

(*a*) Medical Practitioners:

Licensing Body	*Qualification*	*Abbreviation for Registration*
Faculty of Anaesthetists of South Africa	Fellowship	F.F.A. (S.A.).
University of Stellenbosch	Master of Medicine (Radiological Diagnosis)	M.Med. (Rad. D.), Univ. Stell.

(ii) By the addition of the following further qualifications under the heading:

(*a*) Medical Practitioners:

Licensing Body	*Qualification*	*Abbreviation for Registration*
College of Physicians, Surgeons and Gynaecologists of South Africa	Fellowship of the Faculty of Anaesthetists	F.F.A. (S.A.).
University of Copenhagen	Doctor of Medicine	M.D., Univ. Copenh.
University of Stellenbosch	Master of Medicine (Dermatology)	M.Med. (Derm.), Univ. Stell.
	Master of Medicine (Röntgenological Diagnosis)	M.Med. (Rönt. Diag.), Univ. Stell.

(iii) By the addition of the following further qualifications under the heading:

(*b*) Dentists:

Licensing Body	*Qualification*	*Abbreviation for Registration*
Royal College of Surgeons of England	Diploma in Orthodontics	D.Orth., R.C.S. Eng.

REËLS TEN OPSIGTE VAN DIE REGISTRASIE VAN ADDISIONELE KWALIFIKASIES

Die Minister van Gesondheid het in die uitoefening van die bevoegdheid hom verleen by subartikel (4) van artikel *vier-en-neëntig* van die Wet op Geneeshere, Tandartse en Aptekers, 1928 (Wet no. 13 van 1928), sy goedkeuring geheg aan die volgende wysiging van die reëls deur die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad opgestel kragtens paragraaf (*h*) van subartikel (2) van genoemde artikel van die Wet en afgekondig by Goewermentskennisgewing No. 2440 van 14 Desember 1955, soos gewysig:

(i) Deur die skrapping van die volgende kwalifikasies onder die opskrif: (*a*) Geneeshere:

Eksamenoutoriteit	*Kwalifikasie*	*Afkorting vir Registrasie*
Suid-Afrikaanse Kollege van Interniste	Lidmaatskap	L.K.I. (S.A.)
Suid-Afrikaanse Kollege van Chirurge	Lidmaatskap	L.K.C. (S.A.)
Suid-Afrikaanse Kollege van Verloskundiges en Ginekoloë	Lidmaatskap Diploma in Verloskunde	L.K.V. en G. (S.A.) Dip. Verl. K.V. en G. (S.A.)
Suid-Afrikaanse Fakulteit van Narkotiseurs	Lidmaatskap	L.F.N. (S.A.)
Universiteit van Stellenbosch	Magister in Geneeskunde (Radiologiese Diagnose)	M.Med. (Rad. D.), Univ. Stell.

(ii) Deur die toevoeging van die volgende verdere kwalifikasies onder die opskrif:

(*a*) Geneeshere:

Eksamenoutoriteit	*Kwalifikasie*	*Afkorting vir Registrasie*
Kollege van Interniste van Suid-Afrika	Lidmaatskap	L.K.I. (S.A.)
Kollege van Chirurge van Suid-Afrika	Lidmaatskap	L.K.C. (S.A.)
Kollege van Obstetrici en Ginekoloë van Suid-Afrika	Lidmaatskap Diploma in Verloskunde	L.K.O. en G. (S.A.) Dip. Verl. K.O. en G. (S.A.)
Kollege van Interniste, Chirurge en Ginekoloë van Suid-Afrika	Lidmaatskap van die Fakulteit van Narkotiseurs	L.F.N. (S.A.)
Universiteit van Kopenhagen	Doktor in Geneeskunde	M.D. Univ. Kopenh.
Universiteit van Stellenbosch	Magister in Geneeskunde (Dermatologie) Magister in Geneeskunde (Röntgenologiese Diagnose)	M.Med. (Derm.), Univ. Stell. M.Med. (Rönt. Diag.), Univ. Stell.

(iii) Deur die toevoeging van die volgende verdere kwalifikasies onder die opskrif: (*b*) Tandartse:

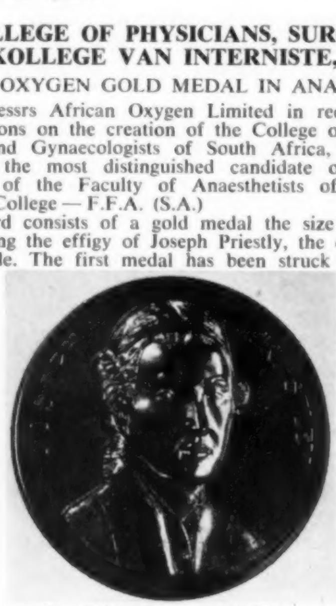
Eksamenoutoriteit	*Kwalifikasie*	*Afkorting vir Registrasie*
„Royal College of Surgeons of England"	Diploma in Ortodonsie	D.Orth., R.C.S. Eng.

COLLEGE OF PHYSICIANS, SURGEONS AND GYNAECOLOGISTS OF SOUTH AFRICA : KOLLEGE VAN INTERNISTE, CHIRURGE EN GINEKOLOË VAN SUID-AFRIKA

AFRICAN OXYGEN GOLD MEDAL IN ANAESTHETICS

In 1957 Messrs African Oxygen Limited in recording their congratulations on the creation of the College of Physicians, Surgeons and Gynaecologists of South Africa, founded an award for the most distinguished candidate obtaining the Fellowship of the Faculty of Anaesthetists of the above-mentioned College — F.F.A. (S.A.)

The award consists of a gold medal the size of a crown piece, bearing the effigy of Joseph Priestly, the discoverer of nitrous oxide. The first medal has been struck and will be awarded in accordance with the following terms laid down for the award:



The African Oxygen Gold Medal

(*a*) The medal is to be known as the 'African Oxygen Gold Medal in Anaesthetics'.

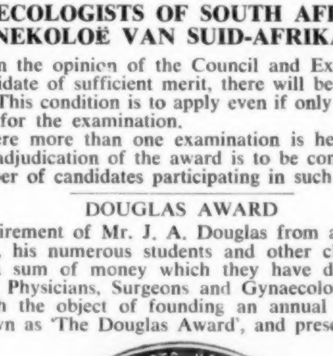
(*b*) The medal is to be awarded to the candidate who, on the recommendation of the Council and Examiners for the Fellowship of the Faculty of Anaesthetists of the College of Physicians, Surgeons and Gynaecologists of South Africa, is deemed the most distinguished in the Final Examination.

(*c*) If, in the opinion of the Council and Examiners, there is no candidate of sufficient merit, there will be no award for that year. This condition is to apply even if only one candidate should sit for the examination.

(*d*) Where more than one examination is held during any one year, adjudication of the award is to be conducted on the total number of candidates participating in such examinations.

DOUGLAS AWARD

On the retirement of Mr. J. A. Douglas from active teaching of surgery, his numerous students and other close associates collected a sum of money which they have donated to the College of Physicians, Surgeons and Gynaecologists of South Africa with the object of founding an annual surgical prize to be known as 'The Douglas Award', and presented annually to the most distinguished Surgical Fellow qualifying during the year.



The Douglas Award

The award consists of a Gold Medal bearing the College Crest, while the reverse will carry the appropriate inscription.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

MULTIFUGIN

Newport Trading Corporation (Pty.) Ltd. announce the introduction of Multifugin, and supply the following information:

Multifugin is a new potent antimycotic and bacteriostatic preparation which is available in liquid, ointment, and powder form.

It contains 5- bromosalicyl-4'-chloranilide and is indicated for the treatment of all forms of dermatomycosis, irrespective of the causative organism. Multifugin is highly effective in the treatment of athlete's foot and ringworm, etc. Bacterial superinfections are equally effectively combated owing to the high bacteriostatic action of Multifugin. Most pathogenic staphylococci and streptococci are inhibited at a concentration of 1 : 1,000 000.

Treatment with Multifugin should be suited to the individual; about 2 - 3 applications a day for 3 - 4 weeks are usually sufficient.

Further information may be obtained from Newport Trading Corporation (Pty.) Ltd., P.O. Box 1871, Johannesburg.

UPIXON

Farbenfabriken Bayer A.G., Leverkusen, Germany, wish to announce the introduction of Upixon, a simple and reliable treatment for ascariasis and oxyuriasis, and supply the following information:

Upixon is a pleasantly flavoured piperazine solution. One teaspoonful (=5 c.c.) contains 1,000 mg. piperazine hydrate. Due to this concentration, the period of treatment can be shortened considerably, whilst the effect is more pronounced.

In ascarides a single dose of Upixon is sufficient for the eradication of the worms. In oxyuriasis safe elimination will be achieved after a course of only 4 days.

Dosage

In ascariasis: Adults and children over 6 years of age: a single dose of 3 teaspoons (15 c.c.) after the evening meal; children 3 - 5 years: 2 teaspoons (10 c.c.) after the evening meal; children 1 - 2 years: 1 teaspoon (5 c.c.) after the evening meal.

In oxyuriasis: Adults and children over 6 years: 1 teaspoon (5 c.c.) 3 times a day, after meals; children 3 - 5 years: 1 teaspoon (5 c.c.) twice a day; children 1 - 2 years: ½ teaspoon (2·5 c.c.) twice a day. These dosages should be taken for 4 days.

Side-effects. Upixon is very well tolerated and in the recommended dosage no side-effects have been observed.

Packings. Upixon is available as a flavoured solution of pleasant taste in bottles of 15 c.c. and 60 c.c.

Further information may be obtained from: FBA Pharmaceuticals (S.A.) (Pty.) Ltd., P.O. Box 10233, Johannesburg.

BOOK REVIEWS : BOEKBESPREKINGS

BRONCHOGRAPHY

Bronchography. By C. Dijkstra, M.D. Pp. x + 157. 106 figures. 45s. Oxford: Blackwell Scientific Publications Ltd. 1958.

This book should be in the library of all those interested in the study of diseases of the chest. The first chapter, which deals entirely with the technique of bronchography, merely describes methods that are now generally accepted. The tilting table devised for posturing the patient is, however, a most ingenious apparatus worthy of special note. Local anaesthesia is used to anaesthetize the nose, pharynx and trachea, and a radio-opaque rubber catheter is then inserted through the nose into the trachea. The patient is then postured so as to obtain good filling of all the segments with the opaque medium.

The remaining 6 chapters of the book are devoted to the radiographic appearances of bronchograms in the different forms of chest pathology. It therefore becomes more in the nature of a reference atlas, with good illustrations of the pathological changes associated with the bronchographic appearances. The inclusion of a certain amount of pathology in conjunction with the X-ray and bronchographic appearances helps to complete the picture of the cases described. The detailed pathology is obviously beyond the scope of this book.

This well-presented volume, which is rounded off with a short bibliography, can be recommended to those interested in the subject. W.P.

PERIMETRY

The Essentials of Perimetry. By Howard Reed, M.B., M.S. (Lond.), F.R.C.S. (Eng.), F.R.C.S. (C), F.A.C.S. Pp. 192+xi. Illustrated. 45s. London, New York, Toronto: Oxford University Press 1960.

Most medical practitioners who need to examine visual fields approach the examination with the attitude that here is a chore, more unpleasant than most, which must be got over as quickly as possible. Howard Reed's book does much to dispel this attitude. It is a pity it did not come on the scene earlier.

It is essentially a simple book with simple diagrams. It clearly shows the applied anatomy of the visual pathways; it relates the visual field to this anatomy and with great clarity discusses various types of field defects.

The chapter on glaucoma is particularly well done in discussing the field defects as such, but I doubt if most authorities would agree that field defects are to be found at all in early glaucoma. Leydhecker has shown that something like a decade probably separates the group of very early glaucomas from the group showing the onset of field defects.

Although this book makes no pretence of being detailed, it should be read by anyone who is interested in visual fields, even if only to see how clear and concise a subject can be made. A.I.F.

CORRESPONDENCE : BRIEWERUBRIEK

POLELA HEALTH CENTRE

To the Editor: I would appreciate the courtesy of your columns to tell all old Polela hands that 'Dopey', the Health Centre dog, has died. All who were stationed there under the Training Scheme for Health Personnel appreciated his companionship over the lovely Natal hills.

'Dopey's' lively ranging matched the hope conceived in the destruction of war and given shape by the Gluckman Commission, that a sound health service would be developed in this country. It seems that this is not to be, for with the dog's death comes the news of the closure of the Institute of Family and Community Health (successor to the Training Scheme) and the dispersal of its personnel. Thus will perish the one truly developmental project to stem from the Gluckman Commission's work and the hope and years that so many of us devoted to its maintenance. This project at its inception was backed by the Medical Association and it would be unseemly were its demise not to be noted and regretted in your columns, to which the Institute has often in the past distinguishedly contributed.

D. J. Lapping

Jorrocks' Plaas
Amanzimtoti, Natal
28 December 1960

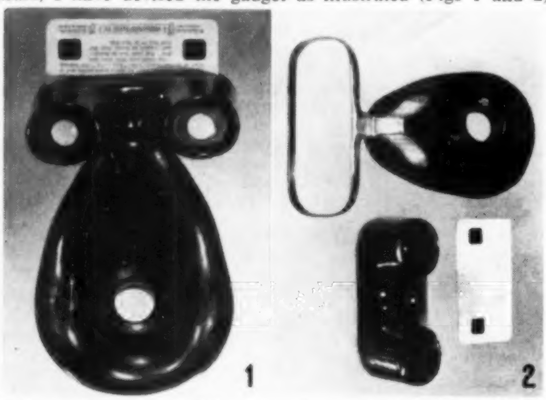
ANAESTHESIA WITHOUT TEARS

To the Editor: The following is a description of a gadget which I have devised for induction of anaesthesia in children.

An anaesthetist attempts to carry out induction of anaesthesia as pleasantly as possible. This applies particularly to children

of the younger age group. For operations of short duration it seems rather a shame to premedicate a child to the point of sleep and so prolong the postoperative period before awakening. To induce by the intravenous method similarly prolongs postoperative unconsciousness and, furthermore, most children are averse to a needle.

To avoid any discomforts and to produce induction without tears, I have devised the gadget as illustrated (Figs 1 and 2).



This consists of a child's face piece to which a bracket is fitted. Into the bracket a Viewmaster is inserted. The face piece with Viewmaster in position is handed to the child who automatically fits the mask to the face by looking into the eyepieces, or the anaesthetist holds the facepiece in position, whichever the child prefers. Slides are inserted into the slot of the Viewmaster and, while the child is looking at the pictures, the gaseous anaesthetic is administered.

I have used this gadget in many cases and find it most satisfying to the patient.

I should like to thank the senior technician of the Orthopaedic Department of the Provincial Hospital, Kimberley, for fitting the bracket to the facepiece.

B. Alexander
Honorary Anaesthetist

Provincial Hospital
Kimberley
6 December 1960

THE SECRETARY'S COLUMN

To the Editor: The Executive Committee of Federal Council is to be congratulated for their foresight in asking Dr. Tonkin to keep the profession informed of 'what is taking place'.[1] I am personally grateful to them for doing this and to Dr. Tonkin for undertaking it.

Nevertheless, it is disappointing to read of the negotiations which have taken place with the insurance companies. I am not being critical of the hard work which I am sure the members of the Executive Committee have put into these negotiations but I do feel that a ceiling income of £2,300 *per annum* is grossly excessive for a medical assistance scheme.

I have no objection to the medical aid scheme, as at present constituted (i.e. an average income of £1,100 *per annum* with a sliding scale of subscriptions while not more than 3% of members earn over £1,750 *per annum*) being extended to all people who qualify, whether they are underwritten by an insurance company or not. This is a fair plan and covers those in the lower income brackets.

It is a far cry, however, from being asked to subsidize all those, whether married or single, earning up to as much as £2,300 a year.

We are not told in Dr. Tonkin's column whether there will be any restrictions to membership. Further information about this would, I am sure, be welcomed. One such qualification is, I believe, an insistence on group membership. When one remembers that two people can constitute a group, this restriction is hardly worth bothering about.

There is another aspect of this matter that we will soon have to face. The two major insurance companies are now in the position where they can take over most of the existing medical aid societies (as is happening slowly) and they can also insure the greater proportion of our population, promising to pay 80% of the fees of a preferential tariff. Over 80% of the population falls in the under £2,300 a year income group; remember too that this is a minimum figure. Many individuals insured is this way will actually be earning more, e.g. there is the question of income from one's wife and children, from dividends, expense account, etc. All this means that we will, in the near future, be dependant on two large insurance companies for the major portion of our group income.

Furthermore we have forfeited our right to assess our own fee and have substituted for this a rigid tariff.

Assume that we wish, at any given time, to adjust the tariff, what then will be our status for negotiation? We are witnessing at present the result of one bout with the insurance companies, at a time when we are not bound to them by any contract. What then, when we depend on them for the major portion of our income?

It would be of value if Dr. Tonkin could inform us of what provisions have been made for negotiations at a later time. After all we have now entered the realm of 'big business'.

All this adds up to one conclusion. We have an important principle to decide upon. Are we to accept the decision of the Executive Committee that the medical profession will, in the future, provide a subsidized service to those in the lower and higher brackets, excluding only the highest income bracket, i.e. the supertax class? After all, most business firms agree that anyone earning over £1,800 a year falls into their higher income brackets.

I am totally opposed to this. I feel that a subsidized medical service should be applied to the lower and lower middle income groups. This is adequately provided for in our present concept of a 'medical aid society'. The upper middle and higher income groups can well afford to pay the difference between their insurance and the private fee. Each of these individuals can then still be assessed for his fee as has been done in the past.

The insurance companies should be made to assist a subsidized medical scheme by introducing more realistic premiums on a sliding scale if necessary and not merely by driving a hard bargain with the medical profession.

Nevertheless, the Executive Committee of our Federal Council have agreed to accept £2,300 as a ceiling. I am sure that they have done this in the true belief that it is in the best interests of the profession, but I am equally sure that many doctors, like myself, will disagree.

I am sure that many will follow the example of Dr. du Plessis,[2] and dissociate themselves from the decision of the Executive Committee. I do not think that this is the correct way to go about it. There are very few doctors who can afford to do this.

Furthermore, it does not adequately bring to the notice of Federal Council the feelings of those that disagree with this decision.

If we are to exercise our democratic right, it behoves each and every practitioner who objects to this, to voice his objection either through the *Journal* or through his Branch Council. If sufficient objections are raised, it obviously cannot be adopted as official policy.

We are at the crossroads and it depends on each and every one of us, which road will be followed.

It would be tragic for our profession if we pursued the wrong road through hasty decisions, apathy, or a lack of courage.

It is the privilege of a majority to decide which path we will follow, but in order to do so, each man among us must speak his mind.

Maurice H. Luntz

308 Dumbarton House
Church Street
Cape Town
29 December 1960

1. The Secretary's Column (1960): S. Afr. Med. J., **34**, 1105 (24 December).
2. Correspondence (1960): *Ibid.*, **34**, 1044 (3 December).

[Some of the points raised in this letter are dealt with in the official announcement on p. 35 — *Editor.*]